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DIVERTICULA OF THE ESOPHAGUS. REVIEW OF THE LITERATURE WITH NOTES ON FOUR CASES OF PULSION DIVERTICULUM OPERATED BY ONE-STAGE TECHNIQUE (MODIFIED GAUB-JACKSON).*

DR. JOSEPH A. PERRONE, Pittsburgh.

From a review of the early literature on esophageal diverticula the condition would seem to be of much more frequent occurrence during the past decade than prior to that time. While the quick lunch counter and the habit of rapid eating that has been acquired by the present generation supplies one of the important etiologic factors in pulsion diverticula, it is probable that the greatest factor in the apparent increase has been the development of Roentgenologic diagnostic technique and the perfection of a method of direct inspection of the hypopharynx and esophagus by a simplified and safe technique of esophagoscopy. Pulsion diverticulum or pharyngeal pouch is usually found without difficulty by a Roentgen examination of the esophagus, using the opaque mixture, while traction diverticulum is frequently not found until an esophagoscopy is done.

This thesis is presented with particular reference to the esophagoscopic phases of diagnosis and treatment of esophageal diverticula.

*This thesis was presented and accepted by the University of Pennsylvania Post-Graduate School as partial fulfillment for the degree of Master of Medical Science.

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History: Records in literature state that Mondiere[1] in 1883 first described the condition clearly. Priority must be claimed for Sir Charles Bell,[2] who in 1816 recorded a case of difficulty in swallowing which was temporarily relieved by the passage of esophageal bougies. The patient died from complications, and at postmortem examination a posteromedian pouch was discovered. He pointed out its pharyngeal origin and also tendered the explanation which to this day is a plausible one. He assumed the sequence of events to be difficulty in swallowing due to a spasmodic contraction of the sphincter at the lower part of the pharynx, hypertrophy and fasciculation of the pharyngeal musculature and, finally, herniation of the mucosa between hypertrophied muscle bundles of the inferior constrictor.

Rokitansky[3] next classified these pouches according to whether they originated primarily from pressure within or traction from without, thus resulting in terms "pulsion" and "traction" diverticula. Zenker[4] in 1877, in his classical study of 27 cases which came to postmortem, gave a true foundation for the symptomatology, diagnosis and morbid anatomy of this condition which bears his name. It is of interest to recall his prediction with regard to the operative possibilities of the condition. "The radical cure of diverticula by surgical operation from without is at present one of our vain wishes, yet we are hopeful that even this operation, conducted on Lister's plan, may at some future day be performed without danger."

The first recorded operation was that by Nicoladini[5] in 1876, when a fistula was established, but the patient died on the sixth day from pneumonia. In 1890 Von Bergmann[6] successfully excised a diverticulum with the formation of a temporary fistula. Kocher[7] performed the first operation in 1892 in which the patient made an uneventful recovery. Since then many successful operations with excellent results have been recorded.

Anatomy: A brief review of the anatomy of the esophagus as a basis for the subsequent discussion would seem in order. The esophagus is a nearly straight membranous muscular tube, being the downward continuation of the food passage beginning at the inferior constrictor muscle of the pharynx. Its origin is from the back of the cricoid cartilage opposite

the sixth cervical vertebra. At the mouth of the esophagus the lower border of the inferior constrictor projects like a mound into the lumen, thus acting as a sphincter.

The security and insecurity of the esophagus depends entirely upon its vertebral bed, *i. e.*, if the vertebral bodies are flat and broad the esophagus rests securely; on the other hand, if the thoracic vertebrae are narrow and pointed the esophagus is quite insecure, and usually falls to the right.

The relations of the esophagus are of utmost importance to both the surgeon and the endoscopist. Behind the esophagus the vertebral column and its anterior wall, in the cervical and upper thoracic portions, joins the trachea in front, forming the party wall. It passes through the posterior mediastinum, and at the fourth thoracic vertebra the arch of the aorta crosses transversely and a vertebra lower, at the fifth thoracic, the left main bronchus makes an oblique line across its front. Below this point the heart lies on it like a weight. In the lower part the right and left pneumogastric nerves are found on the sides of the esophagus and in back of the arch of the aorta the thoracic duct crosses from right to left behind it on the front of the vertebral column.

The esophagus is placed for the most part a little to the left of the middle line of the body. Half way down in its course, at the fourth thoracic vertebra, it swings to the middle line back of the arch of the aorta, but at once goes to the left again and enters the stomach to the left of the spine and in front of the descending aorta at a point placed somewhere between the middle of the tenth and the middle of the eleventh thoracic vertebrae.

Histology: The structure of the esophagus consists of an outer muscular coat of two layers and an inner glandular coat covered with pavement epithelium. These two are joined by a connective tissue layer. The outer layer of the muscular coat consists of longitudinal fibres and inner layer of circular fibres. The ordinary thickness of the esophageal wall is 3 to 4 cm. The longitudinal fibres are attached to the back of the cricoid while the inner layer of the circular muscular fibres is a continuation downward of the fibres of the inferior constrictor muscle.

LENGTH OF THE ESOPHAGUS AT DIFFERENT STAGES — STARK.[8]

Teeth to Cricoid	To Bifurcation
Birth ..... 7 cm. (2¾ in.)	12 cm. (4¾ in.)
1 year ..... 10 cm. (4 in.)	14 cm. (5½ in.)
2 years ..... 10 cm. (4 in.)	15 cm. (6 in.)
5 years ..... 10 cm. (4 in.)	17 cm. (6¾ in.)
10 years ..... 10 cm. (4 in.)	18 cm. (7 in.)
15 years ..... 14 cm. (5½ in.)	23 cm. (9 in.)
Adult ..... 15 cm. (6 in.)	26 cm. (10¼ in.)

To Cardia	Length of Whole Esophagus
18 cm. (6¾ in.)	10 cm. (4 in.)
22 cm. (8¾ in.)	12 cm. (4¾ in.)
23 cm. (9 in.)	13 cm. (5⅛ in.)
26 cm. (10¼ in.)	16 cm. (6⅜ in.)
28 cm. (11 in.)	18 cm. (7 in.)
33 cm. (13 in.)	19 cm. (7½ in.)
40 cm. (15¾ in.)	25 cm. (10 in.)

For memorizing the length of the esophagus at different ages the following approximate figures are given: Birth, 7 in.; five years, 10 in.; 15 years, 13 in.; 25 years or adult, 16 in. (Stark).

DIAMETERS OF TUBES FOR DIFFERENT AGES.

To 8 years	9 mm.
From 9 to 15 years	11 mm.
From 17 years	12 to 14 mm.
Adults	14 mm. (Average)

The esophagus begins 6 in. from the incisor teeth, back of the cricoid cartilage at the sixth cervical vertebrae. It is 10 in. long, and goes through the diaphragm at the tenth thoracic vertebra, 16 in. from the teeth. It is crossed by the arch of the aorta back of the middle of the first piece of the sternum, 10 in. from the teeth. The measurements to be remembered in connection with it are, then, six and 10.

Diameter of the Esophagus: The esophagus is constricted at four points, of which the upper and lower are the most important. The upper one is caused by the cricopharyngeus muscle, the lower by the encircling fibres of the diaphragm. The first, or upper constriction, is a transverse slit slightly less than 1 in. wide, the lower or fourth is apparently of the same width. The long axis of this constriction is from right

to left from behind forward. In addition to these two important constrictions there are two others, second and third, which are often missed unless one watches especially for them. The second constriction corresponds to the arch of the aorta and is found behind the junction of the first and second portions of the sternum and in front of the fourth thoracic vertebra. The third constriction, from above downward, is made by the crossing of the left bronchus in front of the esophagus and this is found at the level of the fifth thoracic vertebra.

DIAMETER OF THE ESOPHAGUS AT THE FOUR CONSTRICTIONS — STARK.

Constriction	Diameter	Vertebrae
1. Cricoid	Transverse 23 mm. (1 in.) Anteroposterior 17 mm. (¾ in.)	Sixth cervical
2. Aortic	Transverse 24 mm. (1 in.) Anteroposterior 19 mm. (¾ in.)	Fourth thoracic
3. Left Bronchus	Transverse 23 mm. (1 in.) Anteroposterior 17 mm. (¾ in.)	Fifth thoracic
4. Diaphragm	Transverse 23 mm. (1 in.) Anteroposterior 23 mm. (1 in.)	Tenth thoracic

Appearance of Normal Esophagus Through the Esophagoscope: The normal mucous membrane of the esophagus under good illumination appears whitish-pink in color. The color changes to red of a varying depth with poor light or inflammation. After trauma the mucous membrane appears edematous. Examination through a small lumened esophagoscope the walls of the esophagus are thrown into large longitudinal folds, these folds indenting the circumference of the central dark area which represents the lumen. Folds are especially numerous behind the cricoid cartilage, making pathological lesions in this area difficult to diagnose. Below the cricoid cartilage and in the cervical portion the lumen is seen to enlarge with inspiration and to close partially during expiration. The esophagus remains ballooned as long as the cricopharyngeus is kept open. As the esophagoscope reaches the first portion of the sternum the pulsation of the arch of the aorta can be seen on the anterior wall. The hiatus of the esophagus is at the level of the diaphragm and appears as a slit or a vasetti. The axis of the opening through the diaphragm is oblique, running from right to left from behind forward. The terminal portion of the esophagus usually shows no lumen

but opens as the tube passes through it. The mucous membrane here is much like that of the stomach, which makes it difficult to tell where the esophagus ends and the stomach begins. The mucous membrane of the stomach is a darker red than that of the esophagus and the longitudinal folds are replaced by the rougie of the stomach.

Types of Diverticula of the Esophagus: 1. Pulsion or pressure. *2.* Traction. *3.* Congenital. *4.* Juvenile. *5.* Double. *6.* Epibronchial. *7.* Epiphrenal.

Pulsion Diverticula: (Pharyngoesophageal Diverticula — Stark or Border Diverticula — Rosenthal[12]). Pulsion diverticula are over 90 per cent of all esophageal diverticula. The majority of cases have occurred in male subjects past middle life, and in a series of cases recorded by Stetton[9] the average age was 54 years. In 27 cases recorded by Zenker there were no females and in the Mayo[10] series the ratio of male to female was four to one. The four cases cited in this paper were of this type, one occurring in a female and three in males. Pulsion diverticulum in the hypopharynx is most amenable to surgical intervention. It occurs constantly in the posterior pharyngeal wall, the entrance is exactly at the border between the pharynx and esophagus behind the cricoid cartilage. The usual location is 17 cm. from the incisors above the cricopharyngeus.

Etiology of Pulsion Diverticulum: Any abnormal pressure from within accompanied with a weak muscular wall will cause a pulsion diverticulum. Jackson[15] states: "It is the barrier presented to the advance of the bolus by the unrelaxed cricopharyngeus that is the functional factor that herniates the pharyngeal wall, thus creating the pharyngeal diverticulum." According to Killian's[14] theory, food insufficiently chewed is capable of disturbing the normal reflex movements which open the esophagus. Slow movements become spastic in nature, resulting in a spastic annual obstruction which holds the larger food particles. If this theory is accepted it must further be assumed that the spasm finally becomes inactive, which accounts for the wide neck seen in some diverticuli. Zenker believes it is a pharyngocele or mucosa hernia, that is, an outpouching of the submucosa and mucosa and its development depends on an anatomic predisposition; for example, the thinness and transverse parallel arrangement of the inferior pharyngeal constrictor fibres. Others assume

there is pharyngeal ectasia or outpouching of the whole wall, this contention being based on some cases in which cross striated muscles were found in the wall of the sac. Other causes which may produce increased pressure from within are forcible quick swallowing, jamming down of large solid morsels and bad dentition. Increased expiratory air pressure, such as sneezing, coughing and blowing of musical instruments, forcing the atrophic muscle fibres to separate or tear.

Weak Triangle of the Pharyngeal Wall: Mosher[6] believes that longitudinal fibres which form the external of the two muscular layers of the esophageal wall split posteriorly and mark off a V-shaped space above the cricopharyngeus muscle. Having separated, the two bundles of the fibres sweep forward and upward around the esophagus laterally to gain an attachment anteriorly to the vertical ridge in the centre of the cricoid cartilage. This leaves the circular fibres of the upper posterior part of the esophageal wall unsupported by longitudinal fibres. This V-shaped gap is about 1 in. long; that is, it equals the height of the cricoid cartilage and is directly behind it. It is through this V-shaped gap that diverticula of the upper esophagus may herniate.

Jackson[28] believes from his repeated observations through the esophagoscope that the orbicular fibres of the inferior constrictor merge with the circular fibres of the esophagus. He has demonstrated that the resistance is on the posterior wall and is due to the orbicular muscular fibres that are essentially different in power and action from either the circular fibres of the esophagus on the one hand, and from the oblique fibres of the inferior constrictor on the other, the oblique fibres remaining contracted while the orbicular fibres relax. With the separate actions of these muscles and the cricopharyngeus muscle acting as a pinchcock, one can well appreciate this anatomic feature as a constant factor in the formation of a diverticula.

Pathologic Anatomy of Pulsion Diverticula: Killian[14] was the first to clearly establish that the pouch was a protrusion of the mucous membrane between the transverse and oblique fibres of the inferior constrictor muscle in the midline posteriorly. This has been confirmed by Goldman,[14] Keith[15] and others. With the increase in size of the pouch it descends and tends to be deflected to one side of the midline, usually to the

left. Downward it will be found behind the esophagus and carotid sheath and usually lies between the prevertebral and pretracheal layers of the cervical fascia. Eventually it may occupy the posterior mediastinum. The sac is usually pyriform in shape and its pharyngeal orifice is, as a rule, moderately wide. The pharynx usually falls into alignment with the sagging down of the sac. As a result of this the upper extremity of the esophagus appears as a narrow aperture on the anterior border of the sac. This readily explains how all food and, likewise, all instruments pass more readily into the diverticulum than into the esophagus.

The wall of the sac varies in thickness. In some cases it is so thin that great care must be taken not to tear the sac, while in other cases it is just the opposite.

Irrespective of the thickness, the constituents of the wall are remarkably constant. The inner lining consists of stratified squamous epithelium. In some cases it may show hyperkeratosis, while in others a definite ulcerative process is noted. In the submucous layer there may or may not be a muscularis mucosa, this being in accord with the variability of the boundary of the esophageal muscularis mucosa. Loose fasciculi of striated muscle arising from the inferior pharyngeal constrictor are usually found around the neck of the proximal part of the sac. The pharyngeal fascia serves as the source for the outer coat or tunica propria. This coat largely determines the thickness of the wall. The loose alveolar tissue which separates the tunica propria from the lining membrane provides a ready line of cleavage and permits of submucous excision.

Appearance of a Pulsion Diverticulum Through the Esophagoscope: With the mere hearing of diverticulum, one unfamiliar with bronschoscopy would immediately interpret that with the passage of the esophagoscope one should encounter an orifice leading from the esophagus proper into the suspected diverticulum; however, this is not true in the greatest majority of cases. The esophagoscope passes directly into the sac dilating the orifice. Many times the orifice will be overhung by a somewhat projecting fold. In other cases one will find the orifice to be very small and rarely steliate. It requires firm anterior pressure with the tube mouth to expose any orifice and it will then be found to be a mere slit and not a gaping orifice. Following a previously swallowed string

and keeping it taut, will prevent entrance of the tube into the neck of the sac and possible perforation of the pouch, thus enabling entrance to the esophagus. When one has located the diverticular orifice, then entry into it with a small esophagoscope will reveal a blind pouch at the bottom of which there is no opening. This sign is invariably diagnostic of pulsion or pressure diverticulum. One must not overlook the fact that in early stages of a diverticulum, a true pouch does not exist. When a pouch does exist, retained liquids and foods are always present.

The color of the mucosa lining of a diverticulum may be reddish or it may be macerated with a grayish color almost resembling an exudate. Minute vessels are plainly seen. There may be superficial erosions and patches of inflammation.

The depth of a pulsion diverticulum is usually from 1 to 4 cm. When filled with food and liquids it may be dilated to a greater extent.

Traction Diverticula: Zenker's definition of a traction diverticulum is one in which the wall of the esophagus has been dragged outward by an exterior force. First observations of this condition were made by Rokitansky and, consequently, it is sometimes known as Rokitansky's diverticulum. They are found mostly in the middle third near the bifurcation where the bronchial lymph nodes lie near the esophagus, rarely in the upper or lower third. The direction of the diverticulum is usually oblique and upwards, rarely in another direction. They are usually situated in the anterior or lateral wall of the esophagus.

Few are ever observed during life and are usually without clinical significance. Seldom do they give rise to any trouble; however, Kraus and Ridder[17] take an opposite view and state that serious possibilities may result from them, the potential danger being perforation with extrusion of food into the mediastinum, lung, pleura or pericardium. Ritter[16] believes that they stand in direct etiologic relation to carcinoma, his opinion being based on the frequency with which malignant disease is found as a result of healed and scarred gastric ulcers.

Etiology: According to Rokitansky, a diverticulum is caused by extension of a perilymphadenitis to the esophageal

walls and the subsequent cicatricial retraction of the granulation tissue. Klebs[18] suggested that some traction diverticula depended on fetal anomalies. Ribbert[19] attempted to show that most traction diverticula were embryonal disturbances, thinking that incomplete separation of the esophagus and trachea left a persistent cord-like union. A muscular defect resulted where the cord was attached and there the mucosa was everted, the adhesions to the inflammatory lymph nodes being secondary. Even if admitted that traction diverticula can develop in this way (but as a rule solitary, Heinen[11]), nevertheless, this interesting theory does not harmonize with the majority of cases which correspond with Zenker's conception, where no such cord is present, only direct fixation by scar tissue. The same opinion is voiced by Hansmann, Brosch, Bevermann and especially Riebold.

Bevermann found in 60 cases that 73 per cent were due to tuberculous inflammation, others to diseases of the lung, pleura and pericardium.

Pathology: The constituents of the wall of the diverticulum depend on the violence of the causative inflammatory process and often the bottom of the pouch consists only of scar tissue or perhaps a shriveled lymphatic gland to which the diverticulum has adhered. The mucosa of the apex of the diverticulum may also contain transported anthracotic pigment. The lymph nodes are chiefly simply anthracotic or softened and shrunken.

Appearance of Traction Diverticulum Through the Esophagoscope: Traction diverticula are very much less likely to be discovered than pulsion diverticula because they are much less in depth and do not present a real pouch. Unless the esophagoscope is kept moving laterally from one side to the other, they may easily escape discovery in the folds of the mucosa, unless a very large esophagoscope is used. Following the location of the diverticulum the subdiverticular lumen is usually easily found. Lateral movements of the esophagoscope will reveal that the esophagus is adherent to some periesophageal mass. The mucosal appearance of the diverticulum is usually the same as in pulsion diverticulum.

Congenital Diverticula: As to whether such a diverticulum actually exists is a question of controversy. Boyd[20] states

that congenital diverticula do not exist. Thorough scanning of the literature revealed only two reported cases in which the writers believed they were dealing with such a condition. Maylard[21] states that they are caused by the persistence of one of the branchial clefts.

Jackson and Shallow[13] reported a case in a child, age 8 years, who, following a rib resection for the evacuation of pus, discharged particles of food through the external chest wound. Due to the poor condition of the child a gastrostomy was performed but no esophagoscopy was attempted. The patient succumbed 14 days after gastrostomy.

At autopsy there was noted a narrow tube springing from the posterolateral wall of the esophagus, which tube opened into the chest. Microscopic examination of this tube revealed three distinct layers, corresponding structurally to the esophagus. This led them to believe they were dealing with an outright congenital diverticulum.

Rush and Stingily[22] reported a case of a child who was born with a swelling on the side of the neck. The child died at the age of 20 days. During this period the swelling increased in size. At autopsy their findings led them to believe they were dealing with a true congenital diverticulum.

Juvenile Diverticula: In this classification[13] would be placed the cases where the diverticulum has its inception during early childhood, manifesting occasionally only trivial symptoms, the symptoms becoming more pronounced during adult life, with increase in size of the sac. It is a possibility that many cases would be classified under this group if a thorough history were taken as to the actual onset of symptoms.

Double Diverticula: Here a number of factors exist as the etiological agents. The esophageal wall may have a number of weak areas with resulting outpouching. Possibility of a diverticulum with leakage and the secondary infection with scar tissue resulting may be the onset of a number of diverticula. They may occur following an old mediastinitis with outward traction; however, these are all hypotheses, *i. e.*, that they are possibilities but not probabilities.

Epibronchial Diverticula: Brosch[23] has described epibronchial pulsion diverticula situated typically on the anterior and left periphery of the esophagus over the left bronchus. These are also known as epibronchial diverticula of Leefegert.

Epiphrenal Diverticula: Deep seated diverticula are mostly found near the esophageal opening in the diaphragm. The orifice is usually a short distance above the diaphragm with the junction of the sac resting upon it. Their origin is supposed to be due to traction and other mechanical factors.

Summary of Symptoms: Most of the symptoms are found only in cases where one is dealing with the pulsion type of diverticulum. Traction diverticulum usually causes no subjective discomforts. The majority of pulsion diverticula present a history of difficulty in swallowing dating back many years; however, on close questioning in the four cases presented in this series, the earliest symptoms presented by the patients have been cough and huskiness of voice; regurgitation of food appeared as a later symptom. This has led the writer to believe that possibly there might be a deranged neuromuscular mechanism of deglutition before a definite pouch is formed; the cough resulting (possibly) from aspiration of small quantities of food or liquids due to reversed peristalysis or spastic contractions of the cricopharyngeus, huskiness of voice resulting from the frequent aspiration of foreign secretions.

When a pouch has formed, the most characteristic symptom is regurgitation of unaltered food and liquids at variable periods after meals. At first the quantity may be small but the tendency is to progressively increase. Another noticeable symptom is an accumulation of saliva in the pharynx, which disturbs rest during the night.

With enlargement of the sac, the initial difficulty in swallowing becomes more pronounced. In some cases the partaking of food is looked forward to with anxiety. In these cases the patient must first fill his pouch before anything passes down the esophagus. Any misadventure may cause regurgitation of all the food eaten. Only by exercising great caution when the pouch has been filled can the patient succeed in swallowing sufficient nourishment to maintain nutrition.

In moderate sized pouches the swelling may be visible on either side of the neck, more often on the left.

Fetid odor to the breath from decomposition of food within the sac and the splashing or gurgling sensation may be annoying. Hoarseness from pressure on recurrent nerve and Hor-

ner's syndrome of unilateral ptosis, myosis, or mydriasis and unilateral flushing, sweating or pallor of the face have been observed.

Boyce's[24] sign can usually be illicited immediately after swallowing. A gurgling sound is produced by pressure of the hand on the side of the neck due to the swallowed air from the pouch.

Clerf[13] has demonstrated with the proper assistance the bubbling of retained secretions within the diverticulum. The patient is examined in the routine manner with a mirror while an assistant makes pressure over the diverticulum, thus forcing the previously swallowed air out of the sac, through the retained secretion.

Diagnosis: The first step in diagnosis should be a thorough Roentgenological study of the case. This examination should consist of a lateral Roentgenogram of the neck, swallowing function consisting of the swallowing of a capsule containing bismuth, and finally, a suspension of barium should be given with the patient in the recumbent position. This method, if performed correctly, gives a complete outline of the entire esophagus. When the diagnosis is made by X-ray, it should then be made positively or negatively with esophagoscopy, thus rendering further diagnostic procedures superfluous.

Jackson[24] states: "To rely upon the radiograph as a diagnosis to the exclusion of esophagoscopy is to take a chance of serious or fatal error." This statement is especially true in the early stages of diverticula when various conditions can be confused with it, such as cardiospasm, malignant or simple stricture of the esophagus.

Preoperative Care: All cases are thoroughly studied before any surgical intervention is attempted. Thorough Roentgenological studies are done in order to determine the exact site of sac and approximate size. A diagnostic esophagoscopy is done in order to rule out any inflammation or new growths within the diverticulum. Twenty-four to 48 hours prior to esophagoscopy the patient is instructed how to swallow a string with a shot. When fluoroscopic examination locates the swallowed shot in the duodenum or ileum, esophagoscopy is then done. It is obvious why a string is used, as any attempt in passing the esophagoscope in a case of diverticulum with-

out a string as a guide may result in serious consequences, in that one would have great difficulty in knowing whether one was in the sac or in the esophagus proper. If in the diverticulum and pressure was resorted to, perforation of the sac would result in a mediastinitis, which would be fatal in the majority of cases. With the swallowed string one could follow it and on reaching the opening to the pouch could enter it and explore the sac thoroughly, then withdraw the esophagoscope — follow the taut string down the esophagus.

Following thorough endoscopic study, complete laboratory studies are performed. A medical consultation is obtained in order to rule out any respiratory or cardiac disease. When all of these are found to be normal or near normal, the patient is then considered ready for surgical intervention. The cases reported in this series have been operated under avertin anesthesia, using 70 to 90 mg. per kilo of body weight, in conjunction with regional infiltration with 0.5 per cent novocain. Preoperative medication is given in accordance with the surgeon's desires.

Treatment of Pulsion Diverticula (Operation of Choice): The choice is between the various two-stage procedures and the one-stage operation with esophagoscopic aid, known as the Gaub-Jackson operation.[25] The advantage of the one-stage resection is that the procedure is shortened to almost half the time. With the esophagoscope the sac is transilluminated and by lifting it from its bed enables the operator to identify it quickly. Esophagoscopic aspiration of retained food and liquids from the diverticulum practically eliminates infection during the operation and the esophagoscope in the esophagus during resection of the sac suturing of its stump, prevents narrowing of the normal esophageal lumen. If there is noted a subdiverticular stenosis it should be treated by the surgeon at the time of operation. Aspiration pneumonia, which may be a postoperative complication, is practically eliminated.

In these cases noted in this paper the one-stage resection with esophagoscopic aid was used. It is suggested that esophagoscopic aid in diagnosis, aspiration of contents of the sac and in transillumination of the sac during its dissection could be used as an aid in the two-stage operation.

Technique of Operation: Under combined avertin and local anesthesia, the patient is prepared in the usual manner. Two

or three days before operation a thread with a three- or four-shot is swallowed as a guide. The operation is never attempted until the shot is located, by Roentgenologic examination, either the duodenum or ileum.

The initial incision is made on the side of which the sac is present, just along the anterior border of the sternocleidomastoid muscle. This should reach from the level of hyoid bone to 1 in. above the sternum and is to pass through the skin and deep fascia, exposing the anterior belly of the omohyoid muscle. This muscle may be divided transversely if necessary. In making this cut, occasionally the external jugular vein may be exposed. If this occurs it should be divided and tied. The common carotid and internal jugular vein is now exposed and should be carefully retraced outward. If an enlarged thyroid gland is noted it may be necessary to cut and ligate the superior thyroid artery. The trachea and the esophagus will be found to be encased in a common sheath, which is to be incised, exposing the esophagus in the posterior part of the wound and the trachea in the front. The trachea is then retracted to the midline.

Following this step the esophagoscope is then introduced. The sac is emptied of its contents by aspiration, to avoid forcing the fluid into the pharynx. The esophagoscope is then introduced to the bottom of the sac; the sac is transilluminated. Rotation of the sac into the wound is done with the esophagoscope. The sac is then grasped and drawn further upward and outward. Sometimes at this point it will be found that the sac is covered with a thin layer of muscle, which should carefully be dissected away. The sac is then freed to the junction of the diverticulum and esophagus, care being taken to free all muscle fibres from its covering. When dissection is complete the esophagoscope is withdrawn from the diverticulum and the proximal end of the swallowed string is threaded through the esophagoscope. The string is pulled taut and the esophagoscope is passed over the string into the subdiverticular esophagus. The sac is then transfixed with a small intestinal needle carrying No. 1 iodized catgut in much the same way as a hernia sac is transfixed. It is then severed close to the esophagus. After transfixation, ligation and amputation, reef the area of guller around the base of the stump by use of three sutures of catgut, inserted at intervals with one picking up the muscular coat of the esophagus. In this

manner inversion of the stump occurs. Simple reef stitches at several points of the circle invaginate the stump easily and firmly, leaving the ends of the reef stitches long enough so that the suture line can be reinforced by the ends carried across and tied to the opposite ends. This narrows the esophagus to a certain extent and if the esophagoscope was not in the esophagus during the suturing, narrowing of the lumen would undoubtedly take place. The musculature upon the sac is then sutured with No. 1 catgut, to give additional protection against leakage. At this stage the esophagoscope is ready to be removed. Prior to doing this a duodenal tube is inserted through the esophagoscope into the stomach and up through one nostril. The incision in the neck is closed in layers. Soft thyroid drains, cellosilk or small split rubber tubes, are placed below the deep fascia.

Surgeons have made slight modifications of the above procedure. The basic principle, however, remains the same in all methods. Recently, Babcock[26] suggested that the initial incision be a transverse one about 7 cm. in length, extending from the midline of the neck to about the middle of the left sternomastoid muscle, directly over the cricoid cartilage. Advantage of this incision is that it leaves an inconspicuous scar, although it gives the operator less room than the conventional incision.

Postoperative Care: The patient is placed in the Fowler's position. Immediately after operation a one quart enema containing 2 or 3 oz. of glucose is given. This is usually retained. Food is given through the duodenal tube every three or four hours, beginning about four hours after operation if there is no nausea. Water and glucose are most preferable. Second day all types of liquids are given, such as orange juice, imperial drink, strained soups, tea, milk, etc.

Oral sepsis is strongly advocated by frequent brushing of the teeth and the frequent rinsing of the mouth with glycothymoline or alcohol, one part to five of water.

All feeding through the tube should continue for at least 12 to 15 days after operation, if the wound is not contaminated and no unforeseen complications have arisen. After the fifteenth day, if the wound is completely healed, soft foods, such as junket, soft boiled eggs, gelatin and thin gruels, may

be given by mouth. Heavier foods are forbidden for at least eight weeks after operation and then thorough mastication and slow eating are emphasized. Prior to the discharging of the patient a fluoroscopic examination of the swallowing function is done for reassurance that no abnormality exists. Drains are usually removed on the second or third day.

Case 1: R. W. C., male, white, age 45 years, railroad worker; admitted May 5, 1932, to the service of Dr. Gabriel Tucker at the Bryn Mawr Hospital complaining of dysphagia, regurgitation of food some time after eating, intermittent huskiness of voice, desire to clear the throat with consciousness of obstruction and pain in the upper chest. These symptoms have persisted for the past four or five years. The only relief gotten is when patient induced vomiting; he then noted food which he had eaten two or three days previously.

Physical Examination: Patient, white, adult, male, well nourished and developed. The physical findings were negative. Clerf sign of diverticulum was negative while the Boyce sign was positive.

Roentgen Examination (Dr. I. P. Nixon), March 30, 1932: "The heart and lungs are negative. The neck is negative. Swallowing function showed a small diverticulum of the esophagus just below the level of the cricoid cartilage on the left side."

Diagnostic Esophagoscopy (Dr. Tucker), May 2, 1932: "The upper esophagus showed a diverticular pouch originating from the pharynx anteriorly just below the level of the cricoid in the left side of pharynx. The inside of the pouch was smooth and presented no evidence of ulceration or infiltration at any point. Following the previously swallowed string through the upper sphincter of the esophagus showing it to be normal."

Laboratory Studies: Revealed negative finding. The pathological report was as follows: "Sections showed the typical structures of an esophageal diverticulum."

Operation (May 26, 1932): The modified Gaub-Jackson operation, as previously described, was performed under avertin and local anesthesia by Dr. Walter Estelle Lee. Patient was given 90 mgm. of avertin per kilo of body weight with morphin sulphate $\frac{1}{4}$ gr. and atrophin sulphate 1/150 gr. as preoperative medication.

Postoperative Follow-up: The patient was fed high caloric liquids through the jutte tube, which was removed on June 9. He was discharged from the hospital on June 14 symptom free. He was seen by Dr. Tucker in January, 1933, was feeling perfectly well, and X-ray examination at that time showed the swallowing function to be normal.

Case 2: F. H., male, white, age 64 years, newspaper man; admitted April 26, 1932, to the service of Dr. Gabriel Tucker at the University of Pennsylvania Hospital complaining of regurgitation of solid foods two to three hours after meals for the past two years. This was preceded by hoarseness and a "rough" feeling in the throat but of no avail. The patient for years has been a hasty eater, does not masticate his food well and is accustomed to drinking hot liquids. On several occasions the patient noted that when he sneezed he would regurgitate food eaten two or three days previously.

Physical Examination: A well nourished male with negative physical findings; neither the Clerf nor the Boyce sign could be elicited.

Roentgen Examination (Dr. J. G. Cohen), April 30: "Swallowing function: Small diverticulum approximately 3 cm. in width, 3 cm. in length and 2 cm. in depth was located just below the level of the cricoid and posterior to the left of the esophagus. The hiatus is probably fairly large because the sac emptied quite readily. Neck: negative. Chest: Fluoroscopic examination of the heart revealed undue pulsation of the beginning of the arch of the aorta and of the left ventricle. Roentgenograms of the chest revealed an elongated arthrosclerotic arch, but there is no aneurysm and no undue compression or deviation of the trachea or esophagus. The heart is slightly enlarged to the left but the chest is otherwise essentially negative.

Diagnostic Esophagoscopy (Dr. Tucker), May 24: "On introduction of the esophagoscope a diverticular pouch was found originating from the hypopharynx and extending back of the cricopharyngeus. It was found to be filled with food. The food was removed and the surface of the pouch inspected. The mucous membrane was smooth and no ulcerations were noted. The string which the patient had previously swallowed was followed and the subdiverticular esophagus explored and found to be normal."

Laboratory Studies: Revealed negative findings. The pathological report was as follows: The sections showed the typical structures of an esophageal diverticulum.

Operation (May 30): The modified Gaub-Jackson operation was performed under avertin and local anesthesia. The patient was given 80 mg. per kilo of body weight. Preoperative medication consisted of morphin sulphate 1/6 gr. and atrophine sulphate 1/150 gr. The operation was performed by Dr. E. L. Eliason.

Postoperative: Patient fed high caloric liquids through the jutte tube. No sedatives were given in order that secretion would be coughed out rather than aspirated into the tracheobronchial tree. The patient made an uneventful recovery and was discharged June 14 with complete relief from symptoms.

Case 3: M. S. H., age 65 years, white, female, housewife; admitted July 25, 1932, to the service of Dr. Gabriel Tucker at the University of Pennsylvania Hospital complaining of regurgitation of food and liquids from one and one-half to three hours after meals, with lodgment of food in the throat of one year duration. This was preceded by a productive cough, which was aggravated in the recumbent position. The cough was productive with sputum which was clear, though frothy, but occasionally became thick yellowish-green. With the onset of the cough there was no pain, dysphagia or choking except when an attempt was made to eat more rapidly than usual. Since the onset of regurgitation of food, the patient experienced a dull burning pain in the epigastrium at irregular periods following the ingestion of food. The discomfort was not relieved by any medications, but only by spontaneous vomiting. Since the onset of the disease the patient has lost 40 pounds.

Physical Examination: Adult, white, female, poorly nourished and markedly underweight. Except for a moderate degree of arteriosclerosis, findings were essentially negative. The Clerf sign could not be elicited while the Boyce sign was positive.

Roentgen Examination (Dr. Pendergrass), July 25: "Large diverticulum of the cervical esophagus."

Diagnostic Esophagoscopy (Dr. Tucker), July 27: "Direct examination showed a diverticulum having its origin just

above the cricopharyngeus with a definite pouch formation in the left side of the pharynx. There was no inflammation of the pouch. The subdiverticular esophagus is normal."

Laboratory Studies: Revealed negative findings. The pathological report was as follows: Gross specimen: The specimen consisted of a diverticulum of the esophagus which measured 3½ cm. in length and 4 cm. in diameter. The inner surface was smooth; there was a small amount of thyroid tissue attached to the surface. Microscopic examination showed typical structures characteristic of a diverticulum.

Operation (Aug. 3): The modified Gaub-Jackson operation was performed under avertin, giving 80 mg. per kilo of body weight. Novocain was used in infiltrating the skin. Preoperative medication consisted of morphin sulphate gr. 1/6 and atrophine sulphate gr. 1/150. The operation was performed by Dr. E. L. Eliason.

Postoperative Follow-up: After operation the patient was fed high caloric liquids through the jutte tube. Sedatives were not given so that patient would cough up secretions instead of aspirating them into the lungs.

The cystitis which the patient had prior to operation was somewhat aggravated, accompanied with urinary retention. This necessitated frequent catheterization of the urinary bladder.

On Aug. 6 the patient suddenly developed auricular fibrillation, which was readily controlled by tincture of digitalis.

The feeding tube was removed on Aug. 11. Soft food was ordered, which food was swallowed with ease.

The patient was discharged on Aug. 17. When last heard from the patient was experiencing no difficulty in swallowing, had gained weight and was feeling perfectly well.

Case 4: A. McN., age 47 years, white, laborer; admitted Aug. 17, 1932, to the service of Dr. Gabriel Tucker at the University of Pennsylvania Hospital complaining of difficulty in swallowing with lodgment of solid food in the throat and hoarseness of voice of three months' duration. For many years the patient had noticed that small objects, like pills, would lodge in the throat for a short time and then pass gradually

down. Since onset of this condition the patient noticed that on arising in the mornings his pillow would be moist from liquids and small particles of food.

Physical Examination: Well nourished but extremely nervous male, whose physical findings were negative. Neither the Clerf nor Boyce signs were elicited.

Roentgen Examination (Dr. Karl Kornblum), Aug. 8: "The heart and lungs are negative. The neck is negative. Swallowing function showed a small diverticulum of the esophagus above the suprasternal notch."

Diagnostic Esophagoscopy (Dr. Tucker), Aug. 12: "Direct examination showed a small diverticulum having its origin just above the cricopharyngeus, with a very definite pouch formation. There were no inflammatory changes of the mucosa of the pouch. Following the previously swallowed string the subdiverticular esophagus was normal."

Laboratory Studies were essentially negative. Pathological report was as follows: "Sections showed an esophageal diverticulum."

Operation (Aug. 17): The modified Gaub-Jackson operation was performed under avertin and local anesthesia by Dr. E. L. Eliason.

Postoperative Follow-up: The patient was fed high caloric liquids and no sedatives given. He made an uneventful recovery and was discharged Aug. 28 completely free of all symptoms.

CONCLUSIONS.

1. Esophageal diverticula are not more common today than a few decades ago, the apparent increase in number being principally due to improved Roentgenologic and esophagoscopic technique.

2. In all cases presenting esophageal symptoms there should first be made thorough Roentgenologic studies consisting of swallowing function and films with an opaque mixture. If a Roentgenologic diagnosis of diverticula is made it should be confirmed by esophagoscopy performed by a trained esophagoscopist prior to the excision of the diverticula.

3. In esophageal diverticula esophagoscopy will: *a.* Give positive evidence of the size and location of the sac. *b.* Note the condition of the sac itself as to whether any inflammatory processes, new growths or ulcerations are present. *c.* Reveal the condition of the subdiverticular esophagus and exclude the presence of stenosis ulcerative neoplasm or other lesions.

4. When an uncomplicated and operable diverticulum is found, and the physical condition of the patient is satisfactory, operative removal of the sac is to be considered.

5. The Gaub-Jackson operation with aid of the esophagoscope is the procedure of choice because: *a.* The time of operation is greatly shortened. *b.* By transillumination of the sac it is more easily found and dissection greatly simplified.

6. In the postoperative care of esophageal diverticula, a feeding tube introduced through the esophagoscope prior to its withdrawal after the sac has been excised is essential. The avoidance of morphin and other cough sedatives is essential in order to prevent pulmonary complications, due to possible aspiration of salivary secretions while the patient is not permitted to swallow.

7. Four cases were reported in this paper.

The writer wishes to thank Dr. Gabriel Tucker, of Philadelphia, Pa., for the privilege of reporting these cases and for his many helpful and constructive suggestions.

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DRY-ICE (CARBON DIOXIDE ICE) BURN OF THROAT.*

DR. PHILIP S. STOUT, Philadelphia.

Report of Case: Earle C., age 15 years, white, American, school boy, average weight, height and intelligence.

History of accident is as follows: On July 31, 1936, at 6:30 P. M., while playing in the school yard, a man came along selling ice cream that was kept cold with dry ice. It appears that the youngsters have a habit of asking for pieces of dry ice, which they put in their mouths and blow their breath over and in this way blow steam from their mouths. Somehow the piece of dry ice that he put in his mouth slipped down his throat. He immediately began to choke, cough, gag and finally vomited, which brought out the dry ice. His throat pained him a great deal; in fact, it felt as if something was burning "like fire" in his throat. His face became red and swollen and for some time the face remained congested in appearance. He complained bitterly about his throat. He went home at once and his parents gave him olive oil, white of eggs and milk, but he could hardly swallow. He was taken to Dr. N. A. Karakashian, who found some evidence of congested throat; however, the pain and difficulty in swallowing was much greater than the appearance of the throat would indicate. His face was swollen and congested. He was unable to swallow his own saliva, and the pain was excruciating.

On the day I saw him, three days after the accident, Dr. Karakashian having referred him to me, he still had considerable pain in his throat and much trouble swallowing; he was still on a liquid diet, and his face was still slightly swollen in appearance. Examination of the throat showed a slightly mottled appearance but no ulceration, blebs, or blisters. At a second examination the day following with a direct laryngoscope, a slight pallor was noted on the posterior wall down near the larynx. Incidentally, it was still very painful for him

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to swallow and he complained of pain during the examination. This painful swallowing lasted about a week. During this time he had to have some sedative to control the pain. He finally got well.

Carbon dioxide ice is now commonly used because it is easily handled and comparatively cheap — about three cents a pound. It is —65° to —70° C., about —110° Fahrenheit. It is used to keep things cold. It is also used in the manufacture of golf balls, freezing the core in the center of the ball. Five claims by workers have been filed because of neuritis of hands and arms. It can easily destroy the skin, as has been shown by its use in skin treatments. Cancer has been produced in mice by the application of CO_2 snow. This was reported in 1931 in a medical journal from the Netherlands. Incidentally, it was used for the treatment of skin cancer about a quarter of a century ago. It was used for many other skin conditions with variable success.

We have looked into the literature, and so far we have not found a similar case.

I wish to thank Dr. Viehoever, Dr. Karakashian and E. S. Moore for their interest in looking up the literature and their general interest manifested in this case.

269 South 19th Street.

OTO-RHINO-LARYNGOLOGICAL COURSE.

University Bordeaux, France, intensive five weeks' course in Oto-Rhino-Laryngology will begin July 26, 1937. For particulars address Dr. James A. Flynn, 1511 Rhode Island avenue, N. W., Washington, D. C.

PASSIVE MASKING AS AN AID IN THE USE OF THE AUDIOMETER.

DR. CHARLES FIRESTONE, Seattle.

Dr. E. G. Witting, writing in the October, 1936, issue of THE LARYNGOSCOPE under the heading "Selection and Operation of Audiometers," gives some pertinent and valuable advice on the subject. It is to be regretted that Dr. Witting's discussion did not appear in an earlier issue, since more and more otologists have in the recent past equipped themselves with this instrument. Like this writer, it is assumed that a good many of these otologists who bought these audiometers encountered some of the difficulties discussed in Dr. Witting's manuscript. The latter succeeds in elucidating some of the obstacles that plague the otologists when using this instrument.

Dr. Witting goes into much detail on the subject of masking when testing with the bone conduction receiver. In the experience of this writer, masking is just as necessary when testing with the higher frequencies of air conduction, as it is with bone conduction. This writer encountered what appeared to him to be insurmountable difficulties in testing for higher frequencies in both air and bone conduction. Patients found it difficult to differentiate between the sound waves reaching them via the receiver and those reaching them by air from the instrument. Several of the masking implements devised by the various manufacturers of audiometers, and others improvised by this writer and not mentioned in Dr. Witting's paper, were tried without availing satisfactory results. In effect, some of the so-called masking aids proved more confusing to the patient than the use of the audiometer without any masking aid. Necessity has long been acclaimed as the mother of invention, and it was out of sheer desperation with the instrument that this writer chanced upon a very simple method of passive masking.

The method is very simple and completely throws into oblivion all the active masking aids devised by the various

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manufacturers. The difficulty of interference is overcome by having the patient in one room, and the audiometer in the room adjoining, so that the wall intervening prohibits the patient tested from receiving by air conduction sound waves emanating from the instrument direct. This writer has found this method practical and satisfactory, inasmuch as the patient informs the examiner by means of signals that he controls from his push-button switch. Practically all modern audiometers are equipped with such an electrical signalling device.

One other source of difficulty encountered by this writer was eliminated by this procedure, and that is the click produced by the cut-off push-button switch operated by the examiner. The patient examined associated this click with the cessation of sound waves. It constituted a psychological interference for him in that it disrupted his attention, and he soon took it as a signal that the examiner had shut the sound off and acted accordingly. This procedure obviates this and allows the examinee more complete concentration on his task. He just does not hear the click.

The Western Electric 6A Audiometer is equipped with a phone so that the examiner may even give the patient instructions from time to time. This writer, however, finds it unnecessary. Otologists thus equipped may use the phone with this procedure. All that is necessary is the boring of channels through the wall for the passage of the wires and connections. Only one silent room as heretofore is necessary, since the audiometer itself is not affected by being in the room next door to the patient examined.

Conclusion: This author finds that this simple method of having the patient in one room and the instrument in the room adjoining eliminates the need of expensive, cumbersome and rather questionable masking apparatus. It has in his hands yielded more dependable audiometric findings.

1422 Medical and Dental Building.

TOXIC LABYRINTHITIS OF NASOPHARYNGEAL ORIGIN.*

DR. THOMAS P. O'CONNOR, Chicago.

The term toxic labyrinthitis is now generally recognized as applying to those cases of true labyrinthine disturbance which are toxic in origin rather than organic. The condition, when it is of infectious origin, is secondary to a remotely situated focus. Clinically, the symptoms are similar to those of the Ménière syndrome with the exception that the hearing is not permanently affected. The patient has sudden, intermittent attacks of vertigo accompanied by tinnitus, nausea and vomiting.

McMurray[1] and Northington[2] have reported cases of toxic labyrinthitis which have responded to eradication of existing foci of infection. Inflammatory processes in the teeth, tonsils, sinuses, duodenal ulcers and gall bladder are regarded as possible provocative sites for the production of this condition.

THE NASOPHARYNX AS THE TOXIC FOCUS.

It is the purpose of this preliminary communication to establish the intoxication caused by nasopharyngitis as an etiologic factor in toxic labyrinthitis. Twenty-three cases have been studied which displayed the typical symptoms of true labyrinthine disturbance. In none of these patients could local lesions of the labyrinth, brain, middle ear, or mastoid be demonstrated; syphilis and poisoning were conclusively ruled out. A search for a focus of infection in every case revealed, in the absence of all other significant physical findings, active subacute or chronic nasopharyngitis. The inflammatory nature of the condition was confirmed by cultures of the nasopharynx in all of the cases, most of which produced a heavy growth of streptococcus viridans. Treatment of the nasopharyngeal vault was followed by prompt relief of symptoms in every case. It is our opinion that the reason for this

*From the Department of Otolaryngology, Wesley Memorial Hospital.

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quick response is that absorption of toxin is immediately inhibited by treatment. In an infected nasopharynx the membrane is usually found to be covered by a thin layer of mucus which has a consistency similar to that of mucilage. Removal of this sheet of mucus and contraction of the lymph follicles doubtless retards the absorption of toxin long before the infecting organisms are killed. Several treatments are usually necessary before the area is entirely cleared of inflammation.

CASE REPORTS.

Case 1: L. B., a social worker, age 45 years, suddenly experienced dizziness two hours after the evening meal. The condition continued for two days without cessation, subsided for two days, and then recurred. During the height of the attack the patient experienced nausea and vomited several times. The vision was blurred and on two occasions she fell to the left against a wall. Phenobarbital, chondroitin and magnesium citrate were taken by the patient with no relief. She was seen by us two weeks after the onset of the condition. Physical and neurologic examination, as well as the history, failed to reveal the etiology of the vertigo. The Graham-Cole test and the gastric analysis were normal. The Wassermann and Kahn tests were negative; a complete blood and urine evaluation showed no deviation from normal.

Examination of the eyes, ears, nose and throat revealed normal visual fields and ocular fundi, normal structures of the nose except for a slight deviation of the septum, clear and equal illumination of the sinuses, no tonsils and normal eardrums. The nasopharynx was markedly injected and the mucous membrane was covered by a thin layer of tenacious mucus. A definite nystagmus was obtained. Culture of the nasopharynx produced a growth of Streptococcus viridans.

The infected area was treated by topical application of appropriate germicidal agents. Relief of all symptoms was obtained in seven days, coincident with the disappearance of all local inflammatory signs in the nasopharynx.

The patient experienced a similar attack 14 months later which subsided in 24 hours, following the above therapy.

Case 2: I. T., an interne, age 25 years, was admitted to Wesley Memorial Hospital because of vertigo and nausea. He

had been conscious of soreness of the parotid gland for about a month and had known that his white blood count was about 13,000. Two weeks before admission he experienced an attack of dizziness which lasted for only two hours. Four days before admission he cut his finger while removing a cast and fainted. Three days later he was overcome by severe dizziness while in the operating room. The patient felt as though he were moving, and closing his eyes did not lessen the sensation. The vertigo increased in severity and 24 hours later he was admitted to the hospital. Neurologic examination revealed no evidence of intracranial involvement. The general physical examination was negative and was confirmed by the following laboratory tests: Gall bladder X-rays normal; Wassermann and Kahn tests negative; electrocardiogram normal; urinalysis normal; red blood count 5,100,000, hemoglobin 15.5 gm., 92 per cent; white blood count 7,150; blood sugar 85.1 mgm. per 100 cc.; uric acid 4.4 mgm. per 100 cc. There was no evidence of pathologic change in X-rays of the nasal accessory sinuses and mastoids. A diagnosis of toxic labyrinthitis was made and he was referred to the ear, nose and throat department for further examination.

The nasal septum was irregular, but not obstructive. There was no evidence of sinus infection. Eardrums were dull and not injected. The tonsils had been cleanly removed. A lateral nystagmus was present. The nasopharynx showed a diffuse, subacute infection, local treatment of which was followed by prompt relief.

Six months later the patient had a recurrence of vertigo with nausea and nystagmus. Again the physical findings were essentially negative except for nasopharyngitis. Treatment of this area resulted in immediate relief of the labyrinthine symptoms.

Case 3: R. R., a nurse, age 20 years, went on duty experiencing severe dizziness. During the next five hours she vomited eight times, displayed a tendency to reach to the right, and by the end of that time was unable to stand. She gave a history of three dizzy spells of increasing severity in the previous eight months. Physical and neurologic examination revealed no evidence of intracranial pathology; temperature 99-100° F.; white blood count 10,950; vision in each eye 25/15; fields of vision normal for form and color; normal fundi; latent diplopia; normal nasal structures; no evidence

of sinus infection; a persistent nystagmus; and a diffuse subacute nasopharyngitis. The tonsils were absent. Culture of the nasopharynx showed Streptococcus viridans. Complete relief of all symptoms was obtained within 24 hours after treatment of the nasopharynx was begun.

During the subsequent two years the patient has had several mild attacks of vertigo which have been immediately and completely relieved by treatment of the nasopharynx. She has suffered no further disability.

Case 4: J. C., employee in the surface lines wrecking crew, age 29 years, was troubled with vertigo, headache and fatigue nine years ago. At that time physical examination was essentially negative except for infected tonsils. After removal of the tonsils he experienced no more attacks of dizziness for eight years. Recently while at work on the tower of the wrecking wagon he became dizzy and was forced to leave his work lest he fall or touch dangerous high tension wires. He experienced several such attacks at frequent intervals before presenting himself for examination.

Again the general physical examination was negative; there was no evidence in the history to suggest drug or alcohol poisoning. Blood count and urinalysis were entirely normal. Nose, sinuses, larynx and ears showed no abnormalities. The nasopharyngeal vault was so intensely swollen and inflamed that it bled after gentle swabbing. A culture from the area produced Streptococcus viridans, Streptococcus hemolyticus and Neisseria catarrhalis. After two local treatments the vertigo and headache subsided. He has had no further trouble.

Case 5: E. G., age 25 years, lost consciousness while sitting quietly in a chair. She was placed in bed, and upon regaining consciousness, gave a history of vertigo, tinnitus, nausea and vomiting of three days' duration. The dizziness persisted when the patient was lying in bed. Neurological examination revealed no evidence of intracranial involvement. The general physical examination and history gave no indication of the cause of the vertigo. Ear, nose and throat examination showed normal structures of the nose; normal sinuses; intensely injected and edematous nasopharynx; acutely inflamed lateral wall of the pharynx; tonsils completely removed. A culture from the nasopharynx produced a heavy growth of Streptococcus viridans and Neisseria catarrhalis. Twenty-four hours

after treatment was begun, the patient was feeling much better. Three days later she had no complaints.

SUMMARY.

Five cases, representative of a series of 23, are presented, which display the typical symptoms of labyrinthine irritation in the absence of any suppurative process in the brain, middle ear, inner ear or mastoid. Common to each was a subacute or chronic nasopharyngitis that harbored Streptococcus viridans. Treatment and eradication of this focus was followed by prompt subsidence of all symptoms.

The manner in which an infection of the nasopharyngeal mucous membrane produces labyrinthine symptoms is a fruitful field for conjecture. The most likely explanation appears to be an absorption into the blood stream of the toxins elaborated from the infection and a secondary irritation or stimulation of the labyrinthine structures. The proximity of the involved area to the Eustachian tube and the middle ear appears to be merely coincidental. Further investigation of this point and into the possibility of any specificity of the offending organisms is contemplated.

CONCLUSION.

Subacute or chronic nasopharyngitis may be the etiologic factor in the production of toxic labyrinthitis.

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30 North Michigan Avenue.

NEW INSTRUMENTS: I. EPISTAXIS CLAMP. II. DOUBLE TONSIL HEMOSTAT.

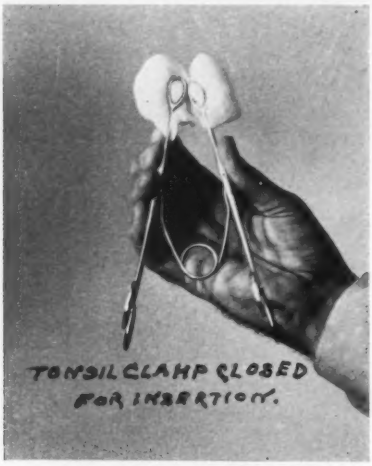
DR. HARRIS D. NEWKIRK, Anaheim, Calif.

I. EPISTAXIS CLAMP.

I submit herewith a sketch of an epistaxis clamp which I made for use in my own practice and which I use, mainly for children.

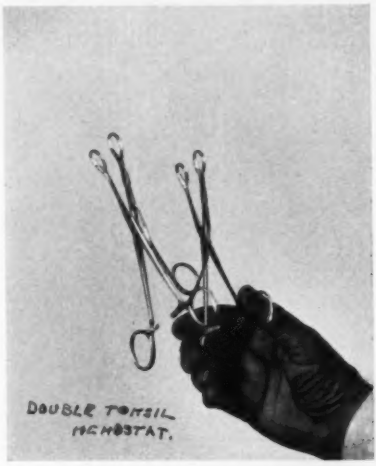
The usual bleeding point is the same; namely, on either side of the septum about the center and several mm. back of the skin line on the mucous membrane. I devised this clamp because of the fact that frequently I was called on at night to stop nose bleeds in children. Parents are very glad to see the doctor arrive and are pleased when he tells them he will stop the nose bleed in a few minutes. He proceeds to do this with a little pack of adrenalin. However, when he picks up his instruments and starts to leave, the parents become apprehensive lest the same thing recur after he has gone. So he is urged to stay and see how it goes. If he leaves, he may be called back before morning.

This little contrivance is made of spring steel (piano wire). A small swab of cotton saturated with adrenalin, or other vein constricting drug, or powdered with a little alum is placed over the bleeding vein. The little gadget then is simply spread



apart and pushed into the nose, one ring on either side of the septum and allowed to spring together. The ring completely encircles the bleeding area and has just enough force to control the hemorrhage without damage to the mucous mem-

branes. It is very difficult to cauterize the bleeding area in a young child, especially while the nose is bleeding. With this instrument, one can control the hemorrhage at once, and cauterize at his convenience. The beauty of it is that the parent, when instructed by the doctor, can use this instrument safely and effectively.



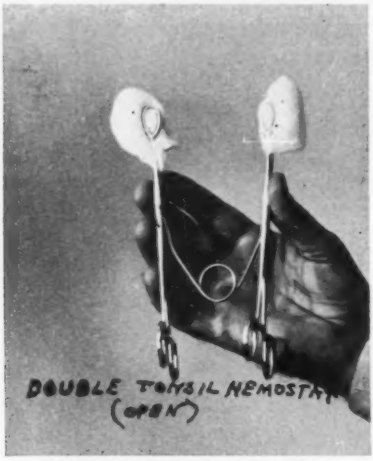
II. DOUBLE TONSIL HEMOSTAT.

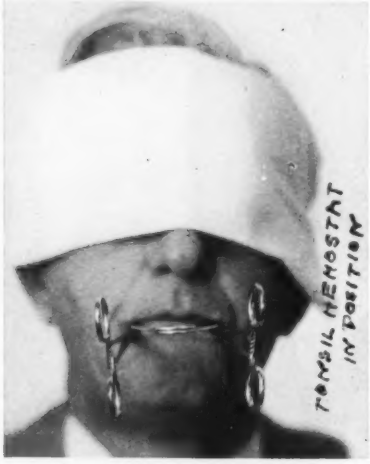
I find this double tonsil hemostat very effective, especially when used following local tonsillectomy where a small amount of adrenalin or other drug has been used to provide a dry field during the operation. Tying blood vessels is at times difficult since the knots may slip, and the procedure at best a little awkward, as in cases of high blood pressure and full-blooded people who are apt to ooze from the whole surface.

This clamp does not constrict the large vessels in the neck by pressure from without or within, but the sponge attachments neatly fit into the tonsil sockets and, if slightly greased before being placed, can be removed and leave a very dry socket. I have found it most satisfying, because I can now do a tonsillectomy, put the clamp in, return the patient to bed and have yet to see a case that has caused me a moment's worry.

The construction of this instrument is very simple, in that it consists of merely two hemostats, securely fastened to a

good piece of spring steel. The sponges are inserted on the outside springs with hemostats attached which press together and are so inserted into the tonsil sockets. When the fingers





are taken off, the spring is released, and the instrument then is firmly fixed so that even the patient himself cannot take it out without pushing the springs together again.

Johnston Clinic.

LINGUAL THYROID.

DR. LEWIS T. BUCKMAN, Wilkes-Barre, Pa.

The following charts conclude the article "Lingual Thyroid," by Dr. Lewis T. Buckman, which appeared in the October and November, 1936, issues of THE LARYNGOSCOPE.

SUMMARY OF CASE REPORTS

Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
88 Feldman, I.	1907	F	31	Found at routine autopsy	(1) Under root and (2) base	None. Died of sepsis following cholangitis	5x2x3 cm.
89 MacCallum, W. G., and Fabyan, M.	1907	....	13	Life	Base	Choking Attacks	.....
90 Stirling, R. A.	1907	F	12	2 years	Base	Dysphagia, dysphonia, dyspnea	Rounded swelling, size of small marble, pinkish vascular, semi-elastic
91 Ungermann, E.	1907	M	30	.....	Base	None; growth found in routine autopsy	.....
92 Perkins, C. E.	1908	F	12	2 years	Base	Dysphonia	Size of hen's egg, bluish red, elastic, movable
93 Aleksieyeff	1909	No details		Could not identify the reference.		.....	.....
94 Kappis	1909	F	25	.....	Base	Preparation only presented.	
95 Austoni, A.	1909	F	12	3 months	Base	Dysphonia, dysphagia	Filling pharynx, size of hen's egg, smooth, vascular, hard, elastic
96 Corwin	1909	M	15	Reported in discussion of Freudenthal's case a case he had under observation of thyroid cyst at base of tongue.			
97 Freudenthal, W.	1909	F	21	.....	Base	Fullness in throat	Size of bird's egg, firm
98 Leulier, M. (Sébileau's case)	1909	F	13	.....	Base	Dysphonia, dyspnea	Size of nut, reddish, vascularized, elastic
99 Leulier, M. (Sébileau's case)	1909	F	17	1 year	Base	Dysphonia, hemorrhage	Large mass, vascularized resistant
100 Meurers	1909	F	20	From childhood	Base	Dysphagia, hemorrhage	Gangrenous mucosa, vascularized
101 Stuart-Low, Wm.	1909	F	23	11 years (operated on before)	Base	Dysphonia, desire to swallow	Large, firm, recurrent
102 Vitto-Massei, R.	1909	F	17	1 year	Base	Feeling of foreign body in throat	Size of small nut. Red
103 Gunn	1910	F	18	.....	Base	.....	.....

THRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
.....	Thyroid tissue	.....	.....	Lacking. Usual thyroid vessels atrophic
ndefinite history of thyroid therapy	Nodule 2 mm. in diam. with compensatory hypertrophy	Rudimentary gland masses found at autopsy	Myxedematous idiot	Lacking. No thyroid vessels on either side
Prelim. tracheotomy. Partial removal thru mouth	Thyroid tissue	Recovery	.....	Isthmus lacking. Small lateral lobes found at tracheotomy
.....	Thyroid tissue	Died following operation for tuberculous hip-joint	.....	Absent
Removed thru mouth	Thyroid tissue	Recovery	.....	.....
.....	.....	.....	.....	.....
Removed	Colloid	Recovery	.....	.....
Partial enucleation by mouth	Thyroid tissue	Recovery	Myxedematous	Absent
Smyth later reported it had been removed partially, showing thyroid tissue. Normal thyroid absent.				
.....	Lymphomatous, but author not satisfied.	.....	.....	.....
Thyroid feeding	.....	General improvement on thyroid feeding; tumor not removed	Cretin	Absent
Thyroid feeding	.....	Improvement in general condition and menses	Hemorrhages at menstruation	Absent
Removed in part by galvanic snare. Postoper. thyroid feeding	Thyroid tissue (embryonal)	.....	Backward Myxedematous	Absent
Prelim. laryngotomy. Removed by splitting tongue from tip to base	Thyroid adenoma	Recovery	.....	Present
Part removed by cold snare in two sittings	Thyroid tissue	Recovery	Menses regular from 14	Present
Tracheotomy. Removed	"Carcinomatous thyroid tissue"	No description of sections. He presented an "exquisitely made model"		Absent

SUMMARY OF CASE REPORTS

Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
104 Livi, C.	1910	F	15	18 months	Base	Sensation of foreign body; dysphonia, dysphagia	Firm left side, pedicle, size of small walnut (pedicle unique to date)
105 Rotgans, J.	1910	F	46	From youth	Base sublingual, submaxillary	Dysphonia, dysphagia	Large tumor covers larynx in mirror view
106 Rutgers, M.	1910	F	21	.....	Base	.....	.....
107 Backer, J. P.	1911	F	Middle Years	Many years	Sublingual	Dysphonia, dyspnea	Within the tongue base not altered
108 Badgerow, G. W.	1911	F	17	2 years	Base	Dysphonia	Hemispherical, firm
109 Brentano	1911	F	42	9 years	Left side of tongue	.....	Elastic
110 Brunner, Fr.	1911	F	50	.....	Base and between hyoid and larynx	Dysphagia	Size of a mandarin
111 Davis, H. J.	1911	F	7	.....	Submental	Disfigurement	.....
112 Gellé E., and Bertein, P.	1911	F	18	.....	Base	Hemorrhage from mouth	Size of large nut, vascular
113 Gellé E., and Bertein, P.	1911	F	40	.....	Base	Dysphagia	Size of mandarin, smooth, firm
	Another report in 1909 by the same authors described two cases in female, but not sufficiently in detail to differentiate them from the cases described in 1911: Probably identical.						
114 Ginsberg, S.	1911	F	23	.....	Base	.....	Conical, smooth, vascular, firm
115 Harris, T. J.	1911	F	12	.....	Base and submental	.....	.....
116 Harris, T. J.	1911	F	16	6 months	Base	Dysphagia, dysphonia	Size of small pigeon's egg, globular, vascular
117 Horn	1911	....	....	.....	Base	Dysphonia	1½" "in diam.", firm cystic
118 Henscher	1911			In discussing Brunner's case, he recalled a similar case yet to be operated upon.			
119 Myles	1911			In discussing Harris' case, cited a similar one.			.....
120 Mayo, C. H.	1911	F	46	.....	Base	.....	.....
121 Mayo, C. H.	1911	F	18	6 years	Base	.....	.....

CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
Removed, galvanic snare	Thyroid tissue	Recovery	Menses at 14	Right lobe present
None	.....	.....	No dysfunction	Not palpable
Had removed greater part three years before. Temporary median section of lower jaw	"Malignant adenoma"	No recurrence after 3½ yrs.	No signs of cachexia strumipriva	Absent
None given	.....	.....	.....	Absent
None given	.....	.....	.....	.....
Two operations on tongue (recurrent)	Goitrous structure	Recovery	.....	Apparently normal
Tracheotomy, removed thru mouth	Thyroid tissue	Recovery	Cretin-like	Absent
Removed, followed that night by acute hyperthyroidism, lasted 6 weeks	.....	.....	.....	.....
Removal by mouth	Thyroid tissue	Recovery	.....	Present
Tracheotomy, removed by median transhyoid laryngotomy	Thyroid tissue, one part calcareous	Recovery	.....	.....
Removed	Thyroid tissue	Recovery	.....	Apparently present
None	Section showed thyroid tissue	.....	.....	Median lobe absent. Lateral lobes small
Partial removal	Thyroid tissue	Relief	.....	.....
Partial removal	Thyroid tissue	Relief	.....	.....
.....	.....	.....	.....	.....
.....	.....	.....	.....	.....
Removed	Cystic colloid goitre	Recovery	.....	.....
Removed by temporary section of jaw in midline	.....	Recovery	.....	.....

SUMMARY OF CASE REPORTS

Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
122 Mayo, C. H.	1911	F	63	18 months	Base	.....	.....
123 Nakamura, Y.	1911	M	31	.....	Base	None	Walnut sized, elastic, smooth
124 Schilder, P.	1911	M	5	.....	Base	Died of meningitis following perforation of spina bifida	Persistence of rudimentary pyramidal lobe
125 Schilder, P.	1911	F	1	.....	Base	Died of furunculosis	.....
126 Schilder, P.	1911		Child	.....	Base	No clinical history	Size of cherry
127 Smyth, H. E.	1911	F	25	Unchanged over 3 years	Base	None	Size of hickory nut, painless
128 Smyth, H. E.	1911	F	21	Unchanged after 2 years	Base	Cough	Size of chestnut
129 Smyth, H. E.	1911	F	21	Unchanged after 2 years	Base	.....	Size of hickory nut
130 Spencer, W. G.	1911	F	23	.....	.....	.....	Hemispherical 3 cur. in diam.
131 Strassberg	1911	M	....	.....	Base	.....	Large
132 Fetterolf, Geo.	1912	M	34	.....	Base	Dysphonia, pulmonary tuberculosis	Rounded, vascular
133 Gachet, J.	1912	M	25	.....	Left submental	Dysphagia, dysphonia	Firm, semi-fluctuant
134 Haynes	1912	F	18	.....	Base and sublingual	Dysphonia, constant swallowing motion	Rounded, vascular, tense elastic
135 Schilling, H. J.	1912	F	15	From childhood	Base and sublingual	.....	Smooth, sharply isolated
136 Walther, H. E.	1912	F	40	26 years followed trauma	Submental	Dysphonia, dysphagia, hemorrhage	.....

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
Lower one removed externally, upper one thru mouth	.....	Recovery	.....	.....
Removed galvanic snare	Thyroid tissue	Recovery	.....	.....
.....	Thyroid tissue with remnant of thyroglossal duct	Found at autopsy	.....	Absent
.....	Rest of the ductus lingualis with small thyroid gland follicles	Found at autopsy	Myxedema	Absent
.....	Thyroid gland tissue	He presented only the preparation fixed in formaldehyde	.....	Absent
None	.....	General health good	.....	Not palpable
None	.....	General health good	Had shrunk as she advanced in years	Apparently present
.....	.....	General health good	.....	Not palpable
.....	.....	.....	.....	.....
Symptomatic	.....	Shrinking, no lues!	.....	.....
None	.....	.....	.....	.....
Left lateral, external approach, removal	Thyroid tissue	Recovery	Postoperative myxedema	No left lobe, also lack of development of left internal ear, and asymmetrical atrophy of left inferior maxilla
Removed by median suprahyoid pharyngotomy	Diffuse colloid degeneration	"Stormy convalescence. Dysthyroidism"	.....	Present
Removed	.....	Recurrence in 9 months	.....	Absent
Removed by transverse suprahyoid pharyngotomy, tracheotomy	Thyroid tissue	Recovery. Speech still imperfect	Cretinoid	Absent

SUMMARY OF CASE REPORTS

Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
137 Getzowa	1913			Demonstrated the preparation from Matti's case.			.....
138 Howarth, W. C.	1913	F	....	.....	Base	Dysphagia, dyspnea	.....
139 Matti, H. (Tavel's Case)	1913	F	47	.....	Base	Dysphagia, dyspnea	4x6 cms.
140 Zuckermann, H.	1913	F	41 days	.....	Base	.....	8x7x3 cms. flat
141 Asch, R.	1914	F	28	14 days	Base	Dyspnea	Size of cherry. Smooth, elastic
142 Howarth	1914	F		Cited a second case (female) in his discussion of Spencer.			
143 Lindt, W.	1914	F	23	From childhood	Base	Dyspnea, dysphonia, hemorrhage	Gray-red, hen's egg, smooth, vascular
144 Seligmann	1914	F	6 mo.	.....	Base	.....	Size of cherry pit, firm
145 Spencer, W. G.	1914	F	20	.....	Base	.....	.....
146 Spencer, W. G.	1914	F	20	.....	Base	.....	.....
147 Spencer, W. G.	1914	F	20	.....	Base	.....	.....
148 Wagner, A.	1914	M	52	.....	Base	Hemorrhage, dysphonia	Size of walnut. (larger at autopsy)
149 Seelye, W. K.	1916	F	18	4 years	Base	Hemorrhage	Size and shape of walnut, vascular
150 Seelye, W. K.	1916	F	25	.....	Base	.....	.....
151 Seelye, W. K.	1916	M	42	.....	Base	Hemorrhage	Engorged with blood
152 Wood, F. C.	1916	F	11	"Always"	Base	Dysphonia	Rounded, red, 2 cms. in diam.
153 Wood, F. C.	1916	F	37	.....	Base, second at upper border of thyroid cartilage	.....	Small

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
.....	.....	.....	.....	.....
Removed tracheotomy	Thyroid tissue	.....	No abnormality	No isthmus
Prelim. tracheotomy, subhyoid pharyngotomy	.....	Death from aspiration pneumonia	Cretinoid idiot	Absent
.....	Cystic thyroid tissue	Found at autopsy	Congenital myxedema	Absent
Removed by mouth	Cystic colloid goitre	Postoperative tetany	.....	Present
.....	.....	.....	.....	.....
Transhyoid pharyngotomy	Parenchymatous colloid goitre	Recovery with postoperative myxedema	Postoperative myxedema	Absent
Removed by galvanic snare	.....	.....	.....	.....
Advised against removal	.....	.....	.....	Isthmus lacking
Advised against removal	.....	.....	.....	Isthmus lacking
Advised against removal	.....	.....	.....	Isthmus lacking
Too ill for operation, died	Thyroid tissue	.....	.....	Palpable
Removed, hot wire snare	Thyroid tissue ("Encepholoid carcinoma" which the essayist doubted)	Recovery, no myxedema	Hemorrhage from mouth usually coincident with each menstruation	Absent
Removed dissected out	.....	Postoperative myxedema	.....	.....
Not noted	.....	.....	.....	.....
Removed	Cystic thyroid, with small nest of parathyroid	.....	.....	.....
Removed	Thyroid tissue with parathyroid	.....	.....	.....

SUMMARY OF CASE REPORTS

	Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
154	Banks-Davis, H. J.	1917			"While awaiting admission for operation for a thyrolingual cyst which produced symptoms interfering with deglutition. I advised the patient to paint the cyst twice a day with tincture of iodine. In a fortnight the swelling disappeared, rendering no further treatment necessary."			
155	Beck, J. C.	1917	M	47	3 days	Base	Dyspnea, dysphagia	Smooth, semisolid
156	Beck, J. C.	1917	F	34	Several years	Base	Dyspnea, dysphonia	Flat
157	Strada, F.	1917	F	Child	.....	Base	.....	Externally negative, 5-6 cms. in diam. submocosal
158	Dawson, G. W.	1918	F	56	3 years	Base	Dysphonia, dysphagia	Lobulated
159	Harscha, W. M.	1918	F	39	9 years	Base	Dysphagia, dysphonia	Size of walnut
160	Jorge, J. M., and Layera, J.	1918	F	25	.....	Floor of mouth	.....	.....
161	Jorge and Layera	1918	F	22	7 years	Base	Dysphonia, dysphagia	Size of tangerine, vascular smooth
162	Lenzi	In discussing Jorges' report, he mentions a case in a female, age 35 years, developing myxedema after operation. His case reported in 1905 was age 42 years.						
163	Lack, H. Lambert	1918	F	Adult	.....	Base	.....	Large, smooth mass
164	Whale, H. L.	1918			Reports a case with hemorrhage in discussing Lambert Lack's.			
165	Rabinowitz, M. A.	1918	F	47	4 months	Base	Dysphagia, dysphonia	1.5 in. in diam., vascular, resilient
166	Mayer, O.	1919	F	37	1 year	Base	Dysphagia, dysphonia	Half-globular, walnut sized, smooth, elastic vascular
167	Barnhill, J. F.	1920	F	40	.....	.....	Dysphonia, sensation of choking	Large bluish mass vascular, size of English walnut
168	Rubeli, H.	1920	F	28	From childhood	Submental and base	Dyspnea	Smooth, half-rounded, vascular

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
.....	.....	.....	.....	.....
.....	Thyroid tissue, with abscess formation	Death from hemorrhage and suffocation	Lingual thyroid increased in size after operation for ovarian cysts	.....
Preliminary tracheotomy, pharyngotomy	Thyroid tissue	Recovery	.....	.....
.....	Embryonic thyroid tissue	Found at autopsy	Congenital myxedema	Absent
None given	.....	.....	.....	.....
Preliminary tracheotomy, pharyngotomy. Removed	Thyroid tissue, some bony elements	Well after 8 years	.....	.....
.....	.....	.....	.....	.....
Tracheotomy, partial removal thru mouth	Thyroid tissue	Recovery	Retarded physical development	Absent
.....	.....	.....	.....	.....
None given	.....	.....	.....	.....
.....	.....	.....	.....	.....
Only a small piece removed, put on thyroid feeding	Thyroid adenoma	Had shrunk to half its size in 2 years and patient gained weight	.....	Absent
Biopsy, part removed with galvanic snare	Thyroid tissue	Discharged with diminished tumor, recurrence in 6 years. Major part then removed by median vertical pharyngotomy with tracheotomy	....	Absent
Removed by snare	Thyroid tissue	Recovery	.....	.....
Required interruption of pregnancy	.....	Recovery from dyspnea without removing tumor	Menses at 18. Lingual thyroid enlarged at each menstruation and in pregnancy caused severe dyspnea. Cesarian section produced prompt relief	Absent

SUMMARY OF CASE REPORTS

	Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
169	Köhl, E. (A report of recurrence in Walther's case (1912) altho Köhl regards it as a second tumor.)	1921	F	49	From school-days, dated from trauma beneath the chin	Base	Dyspnea dysphonia, dysphagia, hemorrhage	Walnut sized, gray-red, blending easily
170	Marcondez, C.	1921	F	Young girl	.....	Base	Dysphonia	Size of tangerine, fluctuating
171	Didier, M. G.	1922	F	49	.....	Base	Dysphonia, dyspnea, dysphagia, hemorrhage	Rounded, soft, vascular
172	Doré F. R.	1922	M	45	.....	Right sublingual	Dysphagia	Size of orange, lobular
173	Droesbecque Goormaghtigh	1922	F	23	2 years	Base	Dysphagia, hemorrhage at menses	Rounded, size of hen's egg, vascular, elastic
174	Hartley, N. J.	1922	F	30	10 years	Base	Dysphonia dysphagia, choking sensation	Size and shape of half a marble, firm
175	Hartley, N. J.	1922	F	22	2 years. Began with attack of fever	Base	Hemorrhage	Size of nut, bluish
176	Madier, J., and Thaleimer, M.	1922	F	14	1 year	Base	Dysphonia	2x3 cm. reddish, vascular, fibrocystic
177	Vernet, M., and Castex, P.	1922	F	26	From childhood	Base	Hemorrhage, nasal tone	3x4 cm. reddish, vascular, fibrocystic
178	Zehner, K.	1922	F	30	3 years, following death of husband in war	.....	Hemorrhage, Snoring, reverse flow of liquids thru nose	.....
179	Bell Tause, H.	1923	F	53	Had growth removed from throat 20 years before, present difficulty 1 year	Base	Dysphagia, dyspnea	Brownish, foul smelling mass
180	Lahey, F. H.	1923	F	25	Since birth	Base	Dysphagia	Size of an egg, firm
181	Luks	1923	F	31	.....	Base	.....	Walnut sized
182	Mollis, W. H.	1923			In discussing Bell Tawse' case, referred to a case from whom he had removed a lingual thyroid to be followed by myxedema.			
183	New, G. B.	1923	M	3½ mo.	.....	Base	Dyspnea, dysphagia	Soft, rounded
184-193	New, G. B.				New referred to 10 additional cases in adults whom he had examined, none of whom had had pharyngeal obstruction.			

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
Removal by resection of jaw	Thyroid tissue	.....	.....	Absent
Biopsy	Thyroid tissue	.....	.....	.....
None	.....	.....	Myxedema	Absent
External approach, removal	Thyroid tissue	Recovery	.....	.....
Partial removal thru mouth, tracheotomy	Thyroid tissue	Recovery	Menses at 18. No menstruation for 3 mo. after operation	Absent
Median suprahyoid, pharyngotomy	Thyroid tissue	Recovery, postoperative myxedema	Postoperative myxedema	No isthmus. Lateral lobes not palpable
Removed by T-shaped incision above hyoid	Thyroid tissue	.....	.....	Absent
Removed thru mouth without tracheotomy	Thyroid tissue	Recovery	No dysfunction	No isthmus or left lobe
Preliminary tracheotomy removed thru mouth	Adenomatous thyroid	Recovery	No dysfunction	Left lobe probably absent
Not removed because of danger of myxedema	.....	.....	Enlarged at each menses	None palpable
Removed by combined subhyoid and transhyoid pharyngotomy	Fetal adenoma of thyroid	Recovery	.....	None palpable
Removed thru mouth	.....	.....	Postoperative hypothyroidism	.....
Removed by sublingual pharyngotomy	.....	.....	.....	.....
.....	.....	.....	.....	.....
No treatment	.....	.....	.....	.....
.....	.....	.....	.....	.....

SUMMARY OF CASE REPORTS

	Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
194	Pierre and Wolf	1923	M	46	12 years	Base	Died 48 hours after extirpation, renal insufficiency. Tumor was firm, excapsulated, homogeneous. Histological: "Endocrine tumor of clear cells."	
195	Rebattu	1923	F	41	Many years	Base	Dysphonia, dyspnea	.....
196	Rossteuscher, M.	1923	F	32	2 years	Base	Pain, dyspnea	Walnut sized, smooth, soft
197	Schmiegelow, E.	1923	F	36	.....	Base	.....	Smooth, elastic
198	Urban, K.	1923	F	19	.....	Base	.....	Walnut sized, round, elastic smooth
199	Wohlgemuth,	1923	F	26	Reported a case in discussing Luk's report, in which a submental tumor developed a year after thyroidectomy, removed, diagnosed cystic goitre.			
200	Anglesio, B.	1924	F	32	5 years	Base	Dysphagia, dysphonia, bleeding once at menstruation	Size of walnut, 2 cm. above surface, rounded, vascular
201	Ashhurst, A. P. C. and White, C. Y.	1924	M	56	20 years	Base	.....	.....
202	Calcagno	1924	F	25	.....	Base	None, vomited dark blood	Size of walnut, smooth, firm
203	Krassnig, M.	1924	F	18	About 1 year	Base	Dysphagia, dysphonia, hemorrhage	Apple-size growth, vascular
204	Roegholt, M. N.	1924	F	"Young"	.....	Base	.....	.....
205	Jorge, J. M.	1925		2½ yrs.	5 months	Base	.....	Size of hazelnut, firm, smooth, vascular

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
.....	.....	.....	.....	.....
Removed thru subhyoid pharyngotomy (partially)	Thyroid tissue (adenomatous)	.....	Myxedematous dwarf	Absent
Preliminary tracheotomy, removed thru mouth	Thyroid tissue	Health good 2 years later	.....	Absent (Proved at tracheotomy)
Removed thru submental region	Thyroid tissue	.....	.....	.....
Removed after prior implantation of thyroid tissue	Cystic goitre	Myxedema improved, he thinks, not so much due to implanted glands as to improvement in the remaining lingual thyroid.	Infantile myxedema	Not palpable
.....	.....	.....	.....	.....
Preliminary tracheotomy. Removed only the left half thru the mouth	Thyroid tissue	Recovery	Did not menstruate until 19	No thyroid shadow by Roentgen ray
First operation 20 years before; recurrence after 3 years. Excision 13 years after first appearance by electrocautery	Carcinoma	Improvement under several months observation	.....	.....
.....	.....	.....	.....	Present
Tracheotomy, section of jaw. Implanted part of the lingual tumor in the abdominal muscles. Thyroid feeding	Thyroid tissue	Swelling had disappeared in a year and her condition much improved	Cretinoid idiot	Absent
Extirpated	Colloid thyroid adenoma	.....	Physically and mentally backward. Never menstruated until after the operation	.....
Preliminary tracheotomy, removal thru mouth	Colloidal fluid	.....	.....	.....

SUMMARY OF CASE REPORTS

	Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
206	McEvoy, F. E.	1925	F	17	1 week	Base	Dysphonia	Rounded, smooth, size of English walnut, vascular, firm
207	Harvey, F.	1926	F	26	For months	Base	Dyspnea, dysphagia, hemorrhage	Attached by pedicle
208	Monro, T. K., and Taylor, M. L.	1926	M	23	18 months	Base	Attacks of choking, dysphagia, bleeding	.....
209	Owens, M. J.	1926	F	36	8-9 months	Base and dorsum	Cough, dyspnea, lump in throat	Elevated 1.5 cm.
210	Torrigianni, C. A.	1927	F	22	13 years	Base	Hemorrhage, pharyngolalia, sensation of foreign body	Pigeon's egg, smooth, fleshy
211	Tweedie, A.	1925	F	31	.....	Base	Globular	
	Suprahyoid tumor removed 17 years before. Normal thyroid absent. No operation on lingual.							
212	Bloch, A. and Lemoine	1928	F	37	1 year	Base	Dyspnea	.....
213	Bloch, A.	1928	M	5 mo.	.....	Base	Alarming nocturnal dyspnea	.....
214	Catania	1928	F	32	5 months	Base	Dysphonia, dyspnea, dysphagia	Size of walnut, irregular, pale, hard, fibrous
215	Demuth, F.	1928	M	25	12 years	Base	Dysphonia, dysphagia	Size of pigeon's egg, smooth
216	Mittag, M.	1928	M	15	.....	Base	Hemorrhage	Nut-sized, smooth, firm
217	Cattell, R. B. and Hoover, W. B.	1929	F	62	40 years	Base	Dyspnea	.....
218	Bisi, H.	1930	F	18	3 months	Base	Dysphonia	Size of nut, smooth, vascular, elastic
219	Carnelli, R.	1930	F	18	1 year	Base	Nasal tone, dysphagia	Smooth, vascular, size of olive, elastic

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
Removed by mouth	Thyroid tissue	.....	.....	Normal
Removed thru mouth	.....	Recovery	Cretinoid, had been on thyroid feeding 2 years	Absent
Tracheotomy, splitting of cheek, removal	None made	The operation had been done before he came under care of authors. The postoperative myxedema improvement on thyroid tissue showed it to be thyroid tissue.		.....
Removed by lateral pharyngotomy	Thyroid tissue	No hypothyroidism for 9 months	.....	.....
At age of 12 had attempted removal thwarted by hemorrhage. Thereafter radium and X-ray without improvement		No results stated	None	Absent proved by exploratory incision
At present removal contraindicated by danger of myxedema. Surgical diathermy used. Biopsy before treatment: Normal thyroid. Now: Small follicles with little colloid, increased c-t.				
.....	.....	.....	.....	.....
Incision and curettage done in a hurry to relieve embarrassment	None made	Improvement	.....	.....
Transverse subhyoid pharyngotomy	Basiloma with remnant of thyroid duct	Recovery	.....	.....
Refused	Struma baseos linguae (clinical)	.....	.....	Absent
Third of tumor sloughed away during attack of neuritic angina	Thyroid tissue	Improved on thyroid feeding	Myxedema	Absent
No operation	.....	Death 5 mo. after first examination	Myxedema at autopsy	.....
Extirpation of a third of the tumor. 4 months later obliged to remove in toto	.....	Recovery. Recurrence in 2 months. Myxedema followed second operation. Gradually did not need thyroid feeding and 7 years later was normal.	No dysfunction	Absent
Refused operation	.....	.....		Absent First appeared with onset of menses at 17. Enlarged with each menstruation. Infantile development.

SUMMARY OF CASE REPORTS

	Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
220	Collet, F. J.	1930	F	7	.....	Base	Hemorrhage	Rounded, vascular, size of nut
221	Moulonguet	1930	F	21	6 months	.....	Dysphagia	Size of an orange, pedicle
222	Willis, R. A.	1930	F	58	No abnormality until after pathogenic destruction of the normal gland	Base	Dysphagia ("bronchiogenic carcinoma").	Hemispherical 1 cm. smooth, pale, firm
223	Caderas, J.	1931	M	39	11 years, attributed to gas near Rheims	Base and lateral	Dysphagia, dysphonia, hemorrhage	Lobulated, vascular, ulcerated
224	Heilman, P.	1931	F	39	Found at autopsy. Death from pulmonary embolism following myomectomy. Submerged in musculature of the	.....	.....	..... uterine tongue.
225	Bisi, H.	1932	F	12	.....	Base	Dyspnea (urgent)	.....
226	Grace, R. V., and Weeks, C.	1932	F	25	.....	Base	.....	.....
227	Terracol, R., Sagols, H.	1932	M	17	1 mo.	Base	Hemorrhage	Size of pigeon's egg, flat, red, vascular
228	Ulrich, H. R.	1932	F	14	16 mo.	Base	Dysphonia	Red, smooth, firm
229	Ulrich, H. R.	1932	F	3½	.....	Base	"Clearing her throat"	Red, symmetrical large as end of adult thumb
230	Ziegelman, E. F.	1932	F	48	4 mo.	Base	Dysphagia, lump in throat	2x2 cm. vascular, dimpled
231	Escalier, J. M., and Arana, E. L.	1933	F	35	4 years	Base	Hemoptysis, nasal tone, dysphonia, dysphagia	.....
232	Hill, R. C.	1933	F	9	.....	Base	Sense of choking	Dark red, lobulated, vascular
233	Mesolella	1933	F	18	7 months	Base	Dysphagia, alteration of voice, sensation of foreign body	Size of walnut, rose color. Smooth, sharp borders

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
.....	Thyroid tissue	.....	.....	Present
Removed, galvanic cautery	Thyroid tissue with malignant degeneration	.....	.....	.....
Autopsy	Hyperplasic thyroid tissue	Death from extension of the mass in the neck (carcinoma)	.....	.....
Radiation	A typical thyroid epithelioma	Recovered, no recurrence	.....	.....
.....	.....	.....	.....	.....
Electrocoagulation because of myxedema developing after his first case Had two sittings	.....	Hemorrhage after each sitting. Regression	Basal metabolism after first seance was +13, before second seance —6, 20 days later —8	.....
Removed thru mouth	.....	Recovery	No dysfunction. B. M. R. —26, 4 months after operation	.....
Removed thru mouth	Thyroid adenoma	Recovery	Slight signs of myxedema 4 months later	.....
Neck explored, lingual tumor not disturbed	.....	Attained healthy adult life	Menses began 1 year before. B. M. R. —8	Absent
Neck explored, no other treatment	.....	One year normal in body growth	.....	Absent
Removed thru mouth, excision	Thyroid tissue	Postoperative myxedema, recovery on thyroid feeding	Postoperative myxedema	.....
Removed	Thyroid adenoma	Well after 1 year	.....	.....
.....	Thyroid tissue	.....	.....	Not palpable
Biopsy. Later removal with galvanic snare, followed by electrocoagulation of base	Tubular sarcomatous hemangiomesothelioma with pseudocolloid	.....	.....	Present

SUMMARY OF CASE REPORTS

	Author	Year of Report	Sex	Age	Duration	Location on Tongue	Symptoms	Appearance
234	Miller, R. H.	1933	F	32	4 months	Base	"Lump in throat"	Size of golf ball
235	Puente Duany, N., and Figueras, W.	1933	F	22	5-6 years	Suprahyoid	None	Firm, size of large nut
236	Bishop, F. J.	1934	F	31	.....	Base	.....	Lobulated, vascular, with pedicle
237	Perlman, H. P.	1934	F	43	1 week	Base	Dysphagia, dyspnea	Firm, smooth, round, 1.5 cm. in diam.
238	Perlman, H. P.	1934	M	55	5 years	Base	Dysphagia, hemorrhage, previously dyspnea	Firm
239	Sagols, H.	1934	M	17	1 month	Base	Recurrent hemorrhage feeling of foreign body, dysphagia	Flat, size of pigeon's egg, reddened, vascular, firm
240	Whalen, E. J.	1934	F	24	5 days	Base	Dysphagia, dyspnea	Smooth, firm, vascular, 4 cm. in diam.
241	Hertzler, A. E.	1935	F		I once removed a very large colloid goitre from a patient who had a lingual goitre the size of a hulled walnut — after the removal of the normally situated goitre, the lingual one shrank and has given her no trouble in the 15 years since the operation.			
242	Levi, L. M. and Hankins, F. O.	1935	F	21	5 days	Base	Choking, coughing, pain, dysphagia	Red-brown firm

(CHRONOLOGICAL).

Treatment	Histological Examination	Outcome	Endocrinal Influences	Thyroid in Normal Position
Removed	Thyroid tissue	.....	Myxedema 2 months later	.....
Radium therapy, in hope of reducing size	Biopsy, normal thyroid tissue	Not stated	.....	.....
Removed with tonsil snare	Thyroid tissue	Recovery	B. M. R. +3	Enlarged
Rest, symptomatic	.....	Relieved of symptoms	B. M. R. +18 after rest became normal	Enlarged
Refused	.....	.....	B. M. R. —10	Absent
Removed thru mouth	Vesicular, thyroid adenoma	Recovery	B. M. R. normal	.....
Biopsy, electro-coagulation	Thyroid tissue	Reduced in size with only one intercurrent hemorrhage	B. M. R. normal	No isthmus, probably no lateral lobes
.....	.....	.....	.....	.....
Biopsy, excised by cautery knife. Postoperative tracheotomy	Low grade carcinoma of thyroid tissue	3 months later, myxedema	B. M. R. —30	Not palpable

AMERICAN OTOLOGICAL SOCIETY.

Sixty-Ninth Annual Meeting, May 28 and 29, 1936.

(Continued from October issue.)

Discussion of Report on Bone Conduction. Dr. Isidore Friesner.

DISCUSSION.

DR. J. GORDON WILSON: It is gratifying to see how the younger members of our Society have taken part so successfully in this symposium. I was pleased to hear Dr. Scott-Moncrieff say that at McGill they have not dispensed with the monochord. At times when unable to decide whether a deficiency of hearing following a recent inflammatory middle ear lesion, especially in those beyond middle life, is due wholly to the conducting mechanism or whether the nerve is involved and to what degree, I have found the monochord serviceable. The disadvantages of the monochord are well known; such as the difficulty of getting sounds of uniform intensity and the separation of the rub from the squeak.

The Committee well deserves the thanks of the Society for its report. The simplicity of its statements leaves little doubt regarding the significance of the various errors it uncovers and the means by which these can be rectified. The report contains a few general remarks which might well have been modified thus, "There is abundant evidence that but few otologists make bone conduction tests in conformity with any of the more precise methods that have been described in the literature." Competent otologists are increasing in number and so are those desirous of using more precise methods of testing. To blame otologists for not following authorities is a little beside the mark; for authorities differ, of which there is evidence even in the present report. For instance, Politzer used a 256 fork, not 512; Bezold used two forks, an A (106) and an a^1 (460) fork, his reason being that by this means he could differentiate combined lesions in the nerve and middle ear. I use a 256 fork and find it better than a 512, making sure that the patient differentiates note from vibration. Otologists fall down in the lack of standards to convey their findings to other examiners, in short, a common language.

The whole scheme of testing the acuity of hearing by means of a tuning fork depends on the identification of the time when the vibrations of the fork cease to be appreciated as sound. This is the identification of a subjective sensation, and subjective sensations are not always easy to follow to their minimum—not to mention after-sensations. So one has to depend on the help and intelligence of the patient as well as to guard against the fatigue.

Having had some experience in trying to get a suitable soundproof room, I believe that for the practicing otologist it is at present unattainable. He must be content with a quiet room. It is easy to get a moderately quiet room but when one tries to tell a colleague how quiet the room is, one partly can assist by comparing the time in seconds between air and bone perception in the patient with a normal.

DR. ROBERT SONNENSCHEIN: I have always felt, as emphasized by Dr. Friesner and in the latter part of the remarks by Dr. Wilson, the necessity of recognizing in a practical way the working conditions under which we live. I am just as much in favor as anyone in precision of tests, in accuracy in scien-

tific work but, after all, we are working in a noisy environment when doing these tests, whether the Weber, Schwabach or Rinne. I pointed that out in the modification Dr. Minton and I made of the Rinne test about 15 years ago. Even though, from the physical standpoint, some of these tests may not be perfect from the practical standpoint they are valuable. We know, when done in a certain way (and many of the cases verified as they were by Bezold at postmortem), you can determine the presence of impaired hearing, and the localization of the lesion, namely, whether in the conductive or perception apparatus or a combination of these two.

I am very glad that the Committee emphasizes the objective standard in the performance of the Schwabach test. In my paper before the American Academy of Ophthalmology and Otolaryngology in 1921 and also on other occasions I emphasized the fact that the method of doing the Schwabach test by placing the fork on the vertex or forehead and then comparing the patient's hearing with that of the examiner was fallacious, because you had to know definitely whether the examiner's hearing was normal, shortened or lengthened. By testing 100 or more normal cases at certain ages, and determining how long, on the average, the fork that you used in a certain definite manner is heard, you then simply compare that with the time the patient hears it.

Secondly, with reference to lateralization in the Weber test of course we know (and I emphasized that in 1911 in a long series of cases) that the Weber test is of value only when it agrees with and confirms the other clinical tests. Of course, when you have a change in lateralization, for instance, with an otitis media on the right side and the patient lateralizes to that side and suddenly the lateralization changes to the better hearing ear and you have vertigo, etc., you know you have an extension to the inner ear, namely a labyrinthitis.

Also the report states that the position and the forks used will cause variation in lateralization. I pointed that out in that same paper. Using the three forks, the a-1 (435 d.v.), weighted C-1, which is d-sharp (154 d.v.), and the A-(108 d.v.) forks, we found in from 11 to 15 per cent of cases that it does make a difference whether the fork is placed on the vortex or forehead and, sometimes, which fork is employed.

With reference to Dr. Wilson's statement regarding the fork used by Politzer, the various times I worked in Vienna years ago the favorite fork was the so-called Vienna fork, which had the weight fixed definitely: with the pitch of d-sharp d.v.

So far as resonance is concerned, Dr. Macfarlan spoke about skull resonance and cross-perception. In 1925 Schoen and Goldberger, of Vienna, published several papers in an attempt to determine how much resonance and cross-perception occurred, and controlled it by means of the stethoscope tips in their own ears. As stated here, the patient was hearing the sound by bone conduction and they were hearing it by air conduction. Bezold showed it beautifully by placing the stethoscope of Laennec on an individual's head; when he on whose head the tuning fork was placed no longer heard it, the individual whose ear was at the other end of the stethoscope still heard, proving that air conduction is longer than bone conduction in the normal.

We know with reference to the cross-perception that when you place the tuning fork at any point not in the median line, for example over the parietal region, as the speaker stated, the greater sound is heard on the opposite side, possibly owing to the union of vibrations. It has been shown that for some reason or other when you place the tuning fork outside of the median line the skull divides itself into two portions, the smaller of which is that adjacent to the fork, the larger, the opposite side; therefore, the resonance is greater on the opposite side and the sound is referred to that side.

With reference to soundproof rooms, I agree with Dr. Pierce, Dr. Friesner, Dr. Shambaugh and others, for, as I have stated previously, we are testing patients who live in the same environment that we do. We know that in a noisy room bone conduction is often apparently increased and, in most cases, as

Politzer stated, the prognosis of a prolonged bone conduction is better than a shortened one, because, with the exception of otosclerosis, the prolongation of the bone conduction means a conduction apparatus impairment, which is often not of very great significance. However, in any case, even though in the soundproof room there is no such thing as prolonged bone conduction, we who live in the noisy environment, if we have a relatively quiet room, can make the tests; and from those data, with the history of the patient, the examination of the ear, nose, throat, etc., and the correlation of these facts, make a diagnosis; then you know your prognosis, and know which cases can be treated. But if you do use a relatively quiet room, I think you get all the needed practical data. If you can work in a soundproof room, certainly, it is finer; the more accurate tests you make the better.

PRESIDENT PACKARD: Before the discussion is opened, Dr. Harvey Fletcher, of New York, a member of the Committee, wants to say a few words on something in the Committee's report on the subject of masking.

Report of the Committee on Methods of Testing the Hearing by Bone Conduction. Harvey Fletcher, Ph.D.

I want to say a few words about the report, and I wish to impose upon you by bringing in an audiometer which has a masking device, which may solve some of the problems we are wrestling with in the report.

With reference to the paper by Dr. Shambaugh, I confess I was quite surprised, after hearing his paper and seeing how he used the audiometer, that he came to the conclusion that he could not use the audiometer but would have to use tuning forks. I mean by this that he determined the degree and kind of deafness of his patients by the audiometer and also determined whether their hearing had changed by the audiometer. I do not quite follow how he arrived at the conclusion he did.

I want to emphasize what Mr. Wegel said. It seems to me the question of stimulation by a tuning fork or any other device is a physical problem; you can do it any way, provided you have the same sort of vibration at the same point. One can use tuning forks, if they are used properly, and one must get the same results if one uses them as though using an audiometer. It is only a question of which is more convenient and which is the more flexible. With those who have been using tuning forks and have built up a history and experience with them, it is quite evident that they will want to continue with them. I think that is quite proper.

In my opinion the younger otologists who use the new audiometric methods will forget completely about the tuning forks as time goes on.

The Committee discussed the testing with high and low tones and I know it has been discussed in this Society before, as to its significance. There had been a tendency in the past to place most effort upon testing with very low-pitched tuning forks and very high-pitched tones produced in various ways, such as the Galton whistle, and other devices.

According to the present knowledge as to the mechanism of hearing, including that obtained from animal experimentation that has been going on recently, it is quite evident that that is just the wrong way to do it. Those tones have the least significance of any because, in the high frequency region, the variation from the normal is so large that when you get a loss you do not know whether it is just a variation from the normal or whether it means some lesion. If it were not for that fact, it might have more significance. In the low frequencies, according to our present conception, a 256- or 128-cycle tuning fork or tone will stimulate the basilar membrane right down to the end of it. If there is anything wrong with that or any of the mechanism, it will show up for this frequency as well as any lower frequencies. As you go lower and lower in the frequency, the difficulty of making the test increases. It was the opinion of the Committee that it is not necessary to go below 128; most of the members of the Committee were inclined to say even 256. And also

it is not necessary to test higher than, say, 8,000, although that was not emphasized in the report because everyone did not quite agree to it. If I, as a physicist, were dictating, I would say you would get most of the information about the mechanism by testing only between the limits 200 and 8,000 e.p.s.

Now as to the masking scheme I spoke about, it is attached to the new Western Electric audiometer, which I have here. The effort has been to make this audiometer as simple as possible. Such an effort was also made in the design of the 2A audiometer. But this one, I think, is made even more simple. There are only two dials, the pitch dial and the intensity dial. It works either on A.C. or D.C. lighting current and has no batteries. In order to save Dr. Fowler — who seems to be the only one who makes the important subtraction between bone conduction and air conduction — the task of making such subtractions, we have a dial here. You slip this dial into place and then you can read the bone conduction from the normal directly from the dial. This detachable dial has the advantage that, as time goes on, and if the American Standards Association which is studying this question changes standards for normal, all you have to do is to take this dial off and put on another one to meet the new requirement. This dial reads the same as the 2A audiometer. With this dial on, you can read directly the bone conduction from normal.

This is the masking device that gave me the excuse to present the audiometer because it is on the subject. It makes it possible to produce masking without adding any equipment to the audiometer except a small switch, a coil and some simple resistances. The tone current from the audiometer comes into this circuit and divides into two parts. Part goes into the bone conduction receiver which is held on the bone of the head for making the test, and the other receiver is held on the ear which it is desired to mask.

When the tone comes on you have a continuous tone going in this masked ear, and the circuit is arranged so that the actual amount of stimulation given to it can never get through to the ear being tested, no matter where you turn the dials, because when the sound intensity is sufficient to hear in the tested ear, the sound is never sufficiently high in the masked ear to reach the tested ear. This circuit is arranged so that even though you go through the entire frequency and intensity range, it maintains the relative sound intensities, so that this is still true.

You will say, "How can you tell whether you are hearing in this ear or that one since you are using the same frequency?" You could not tell by listening unless you interrupted one of the tones. You interrupt the testing tone, and if you hear the interruptions you know you are hearing this tone. This little button makes it possible to interrupt the testing tone without interrupting the masking tone. Thus you determine the threshold in the usual way when masking is not used.

DR. ROBERT SONNENSCHEIN: Is it permissible to ask Dr. Fletcher a question regarding that? How do you explain those curves shown by Dr. Shambaugh? (By the way, they were shown before the Chicago Laryngological Society.) How do you explain the fact, with the conduction apparatus impairment in one case, and apparently perception apparatus impairment in the other, you get practically identical curves with reference to bone and air?

DR. HARVEY FLETCHER: I would prefer to refer that to an otologist like Dr. Fowler to answer. I would have to question whether they were the facts, if, as you say, one is perception and the other conduction deafness, and both cases gave the same audiograms for bone and air conduction.

DR. SONNENSCHEIN: Tuning fork reaction, whisper test and every other data you could possibly have, were used.

DR. FLETCHER: I am not an otologist, as you know, but we have made hundreds of tests at the laboratories, both on bone conduction and air conduction. Dr. Fowler made thousands of them. From two complete audiograms, one on air and one on bone conduction, I think you can predict what you will get in every one of the other hearing tests.

DR. EMIL AMBERG: One of the gentlemen spoke about the difference in experiments in a noisy room and in a quiet room. Another one spoke about ideal conditions and somebody spoke about repeated examinations. If you make repeated examinations, there must be doubt about the correctness of some of them. Then somebody said the tests must be made under the same conditions.

I would like to put one little point into the discussion which may, perhaps, not be of much value, and yet, it may be of greater value than we imagine at present, and that is the disposition of the examined person at a given moment.

One of the essayists spoke about fatigue, which is a very important factor. I just want to call your attention to Urbantschitsch's textbook (page 68). You will find very illuminating facts in it. Then in the Encyclopedia of Otology you will find an article on fatigue by Max Goerke.

In experiments which are made in the psychophysical laboratories, great stress is laid on the fact that the experiments should always be made under the same conditions, even, I think, at the same time of the day, because, as Politzer has said, we hear worse at certain times. We know that lack of sleep, hunger and various drugs affect us. Experiments have been made on the vestibular apparatus with drugs. As far as I know, no systematic experiments have been made on the perception apparatus. It is also important that neuroses be taken into consideration.

So, the point I want to make is that in spite of the excellence and the painstaking accuracy of the report which was so kindly sent to us, it might not be amiss in the future to consult with a psychophysicist and use, in the experiments, the same precautions which are carried out in psychological experiments.

DR. MAX A. GOLDSTEIN: There is one problem in this rather perplexing bone conduction question that seems not to have been touched upon by the various essayists. I refer to the relation of tactile impression to sound perception.

Several years preceding Gault's introduction of the teletactor, we tried a practical experiment on congenitally totally deaf pupils at Central Institute. These pupils had been trained, with their finger tips, to recognize pitch variation and pitch differentiation on the sounding board of the piano or organ, and on the amplified loudspeaker.

One boy, age about 10 years, was placed in the gymnasium on a high mattress so as to overcome the possibility of conduction of sound through the boards of the floor. He was stripped to the waist and his hands upraised, and through his skin, evidently, and the tactile possibilities of efferent nerves in the skin, he could differentiate sound frequencies, low, middle and high, either in individual notes or in chords struck on the piano or on the organ. What does that indicate?

Another practical experiment. Congenitally totally deaf children (and they are good experimental material that fits in with this type of observation and investigation) are good subjects for definite results in this bone conduction problem. Congenitally deaf children who had been tested out, and who had been found to have an absolutely negative acoustic and static labyrinth, and, consequently, no peripheral labyrinth, were tested with tuning forks, were tested with the audiometer on any part of the body, and felt those tone differentiations.

That brings to mind the classification of Dr. Guild, in which, either by air or by bone conduction in his diagrammatic representation, he assumes this function to be in the organ of Corti. I do not think we know so much about the organ of Corti yet, but we must not lose sight of that part of the brain cortex that is associated with the central perception of sound.

The congenitally deaf child who has a residuum of hearing is trained in this way. When we check him up on the audiometer and find a small degree

of sound impression in the low frequencies, after we have trained him several years, and put him back on the audiometer, he demonstrates no more hearing, so far as the audiometric record is concerned, than he had when he started.

Lorente de No, at Central Institute, in the last few months, has come to the definite conclusion in making laboratory tests of efferent sound impulses in the comparison between a sound traveling over the sciatic nerve or over the auditory nerve, that the time measurement of that sound perception, centrally, is exactly the same. In other words, the auditory nerve, like the sciatic nerve, is simply an electric conducting wire.

DR. HORACE NEWHART: On this very auspicious occasion, when we have with us so many internationally known acoustic engineers, I wish to ask two questions of practical interest to the practicing otologist.

The first one is regarding the soundproof room. We all know that none of us, or practically none of us, aspires to the possession of an absolutely soundproof room. Many of us, however, are making the attempt to secure an acoustically treated room in which the noise level is so reduced as to make it very practical, by eliminating the most distracting, masking noise which makes reasonable accuracy impossible.

In this report I note the suggestion that it is better to line such rooms with a hard surface rather than with sound-absorbing material. It seems to me that this at once introduces elements of error in making the ordinary tests, in that we get an increased amount of noise from the reverberations of the noises that penetrate within our acoustically insulated space.

The second question is regarding the interposition of a thick cushion of sponge-rubber between the auricle and the air conduction receiver, in testing acuity for air conduction. This necessarily makes a very considerable difference (according to the degree of pressure which the patient makes upon the receiver) in the distance between the vibrating diaphragm of the telephone receiver and the tympanic membrane. It seems to me that we thus introduce an appreciable element of error. I would ask if the advantage from this interposition of a thick layer of sponge-rubber to eliminate bone conduction compensates for the possible element of error thus produced?

DR. HARVEY FLETCHER: Dr. Newhart asked a question. The Committee made a report on the question of soundproof rooms and made a recommendation, as he pointed out, that the inside of the room be constructed with hard walls. I do not think the Committee meant to make it uncomfortably hard, but wanted to point out that it was not necessary to have it completely filled with absorbing material, as has been done in the past.

However, I think there is some advantage in having absorbing material on the inside because it helps to reduce the noise. There is a disadvantage in that it puts the patient in a little different environment, and that is the point the Committee wished to point out.

I desire to say a word about the question of testing in a noisy room and making correction for it. The corrections cannot rightly be made. To illustrate what I mean, consider the following: If I were in a very noisy room with a person who is hard of hearing, I might be considered the patient instead of the hard of hearing person, because I would be more handicapped than he. In some tests you would find that he could hear better than I. That is an exaggerated case, but the same principle holds for all degrees of noise. If there is any noise present, you cannot differentiate between small amounts of deafness and normal. The more noise there is, the larger the range of deafness where no deafness at all can be detected.

DR. STACY R. GUILD: May I answer Dr. Newhart's two questions? Most sponge-rubber earpieces are not satisfactory for the reason he gives. We have used at Baltimore a kind originally selected by Dr. Bunch. Within the last two years I have discovered another satisfactory one. We check the different ones by actually testing normal people, without and with the earpieces, and

find that the use of certain sponge-rubber earpieces on the receiver of the audiometer does not cut down the threshold readings obtained. Selection must be on the basis of actually trying against the normal, until one is found that has the same acoustic impedance as does the receiver applied directly to the ear. Sponge-rubber earpieces do help to exclude extraneous noises.

With respect to the hard walls, I call attention to the wording of the report: "The Committee recommends also that the walls of this room or booth be of hard material, because many patients are so disturbed by the absence of reverberation in a padded room that they do not cooperate well in the tests."

If you have ever been in a completely soundproofed padded room, you will appreciate the feeling of a patient who is not accustomed to it.

CLOSING DISCUSSIONS.

DR. STACY R. GUILD: I wish to question two features of this instrument for masking. First, I do not believe that the use of the same frequency is as good for masking purposes as is a mixture of tones. One reason for this belief is because of the things we are all familiar with from the two-fork test.

However, since this comes from the Bell Laboratories and has the support of Mr. Wegel, Dr. Fletcher and others of their groups who know more about the effect of pure-tone masking than I do, I hesitate to speak further about it at this time. I do feel, however, that we need to do considerable experimenting with that method of masking for clinical purposes before it can be recommended.

The second thing about the instrument shown by Dr. Fletcher is that it maintains a constant differential between the masking intensity and that of the test tone. That is all right for certain types of cases, but for patients with a unilateral conductive lesion, unilateral otosclerosis or a chronic otitis, one needs a very different intensity for masking than when both middle ears are normal. The differential in intensity at which sounds "cross over" from one ear to the other is much affected by such conditions; therefore, a masking apparatus should provide means of changing the intensity of the masking sound with respect to the test sound.

DR. FLETCHER: This is only for bone conduction masking.

DR. GUILD: All masking for clinical purposes must be by air conduction. One cannot use bone conduction for masking because both ears are stimulated. That is in the Committee report, and I am thoroughly convinced that it is so.

I was much interested in Mr. Wegel's comment of a 35 decibel loss when a plane wave front strikes the integument. I did not call attention (it will show in the publication) in my first lantern slide to the arrow from the air side of the chart direct to "integument" and then up along the bone conduction side of the chart. Elaboration of that diagram, with consideration of the coefficients of absorption, and so forth, for soft tissues, should be done by someone with Mr. Wegel's special qualifications. I feel that there is an important "tie-up" between bone and air conduction and that this relationship may explain many things.

DR. DOUGLAS MACFARLAN: I think we would miss a good deal at this meeting if we did not remember Dr. Guild's paper and his description of this discrepancy between AC and BC in the cases found associated with those fractures. I think they will lead us far if we follow them a little further.

As to Dr. Fowler's paper: I think there is need of amplifying the clinical data on BC hearing by the report of the physical analysis of these receivers. I think we oftentimes do not know enough about the physical characteristics of the instruments we are using, and we have very little data on the physical condition of the BC receivers.

DR. GEORGE E. SHAMBAUGH, JR.: Dr. Fletcher criticized me for using the audiometer to compare improvement of hearing when I said it was not worth

anything. I did not say the audiometer is not worth anything for comparing the improvement in hearing. It is more valuable than any other test we have for that. For diagnosis, I do not believe it is as valuable as the tuning fork test.

Take the last patient with the diplacusis and the low-tone nerve deafness. I maintained that with the audiometer curves, no matter how you make them, bone conduction compared with air conduction, you could not diagnose this patient as low-tone nerve deafness. There is a false impression of hearing for the low tones in the standard curve. The patient could not hear the low tones at all by air conduction, as shown by the tuning fork test.

DR. FLETCHER: Was that bone conduction plotted from the dial or have you made subtractions?

DR. SHAMBAUGH: Without subtractions.

DR. FLETCHER: Would the subtraction show what you want?

DR. SHAMBAUGH: No. It would show the Rinne negative, and yet the fork showed the Rinne was positive. I maintain that the audiometer could not have diagnosed this as low-tone nerve deafness. In the ordinary case of deafness, it is easy enough, with the audiometric curves, comparing air and bone, to make the diagnosis of nerve deafness or conduction deafness. It is in these borderline cases that diagnosis is most important and most difficult; Therefore, I think tuning forks are necessary.

Here is the patient with a unilateral dead ear. I know he has a dead ear, for I removed his labyrinth because of suppurative labyrinthitis. The bone conduction curve for the low tones is heard through to the other side. The standard audiogram receiver, I believe, is also heard around through the head to the other side by bone conduction. I believe it is heard through the head by bone conduction from the standard receiver which fits close to the head.

DR. BUNCH: My idea of using the masking was to prevent the hearing in the good hearing ear in cases of complete deafness. If you start at the right ear, this right shows the hearing in the left ear, because you have not masked out the hearing in the left ear.

DR. SHAMBAUGH: That is correct, masking is necessary. Even with masking I think you get false hearing in the bad ear, in case of conduction deafness, because I believe the patient is getting some element of bone conduction from the standard receiver and you are not really measuring the air conduction.

DR. BUNCH: Evidently in this chart you are not using sufficient masking.

DR. SHAMBAUGH: I did not use any masking.

DR. R. SCOTT-MONCRIEFF: As regards the matter of absorption of sound by soft tissues, it may be of interest to say that in one of our cases, the case of cholesteatoma with epilepsy, the patient had had a radical mastoid operation. A day or two after the operation was done, the bone conduction was tested, with the fork placed on the bare bone in one instance, and on the edematous tissue just back of the open wound in the other. There was about three seconds difference in the two, which we thought was rather striking. We felt that there should be very much more difference, due to the edema.

Referred Pain in the Ear. Dr. Perry G. Goldsmith.

The author stated that the subject was being discussed only for the second time since the Society's organization.

The anatomical connections and reflex pathways that may be associated with referred aural pain were reviewed. An attempt was then made to review

systematically the possible lesions that might cause reflex stimuli from the various regions. A number of cases, with short case histories, were presented to illustrate the various points that had been discussed.

DR. GEORGE M. COATES: I shall not attempt to discuss these almost innumerable causes of referred pain in the ear, but simply call attention to one particular cause that he, I think, mentioned, but did not stress.

At the meeting of the American Medical Association in Kansas City a couple of weeks ago, there was an excellent paper there by Dr. James Costen, of St. Louis, on the effects on the ear of closing of the bite. Dr. Costen showed that, frequently, when there is a malocclusion, closing of the bite on one side, due to dental deformities, particularly loss of teeth on that side, the temporomandibular joint becomes subluxated or, at times, eroded, and these erosions and malformations can be shown very well by the X-ray. I have had some experience with this condition.

These erosions, particularly the erosions in the joint from closing the bite, do cause referred pain in the ear. They, also, at times cause conductive type of deafness, and particularly tinnitus aurium.

Dr. Costen's idea, and so has ours been, is to open the bite, and that can be accomplished in a diagnostic way by putting in a couple of small cork discs between the upper and lower jaws on the affected side, to see whether temporary opening of the bite will relieve the symptoms. If there are any signs of improvement in the patient's condition, it is then the task of the dentist to put on "skids" or build up the teeth on that side, so as to equalize the pressure on both sides.

DR. J. GORDON WILSON: If no abnormal condition can be seen by aural examination, information of the source of the referred pain can be obtained from observation of the painful area, for it is obvious from what Dr. Goldsmith has said, that the location of this area will vary with the distribution of the nerve.

One point perhaps not in direct relation to Dr. Goldsmith's paper, but of importance, might be mentioned; namely, the cause of the pain that may arise from a loud noise. Although assumed by some to have a direct relationship to the audiometric curve there is no evidence that it is. Certainly a part is related to painful sensations arising in the membrana tympani, which is fundamentally a protective structure, as I pointed out several years ago, and a part may be due to excessive contraction of the tensor tympani, as Hallpike has recently suggested.

DR. ROBERT SONNENSCHEIN: Probably Dr. Goldsmith mentioned a fact in his paper, but did not have opportunity to read it, and that is the point that in tuberculosis of the larynx, an ulcer on the posterior wall will often cause pain that is referred only to the ear and not to the throat.

DR. HARRIS P. VAIL: There are a few things in Dr. Goldsmith's paper that I might criticize, if I may use that word. One is his failure to give more credit to the great superficial petrosal nerve in the transmission of sensory impulses. I think it has been pretty well shown by anatomists, Dr. Koontz, of St. Louis, Dr. Fenton and others, that the great superficial petrosal nerve is a sensory nerve, in addition to any sympathetic fibres which it may contain. I think this can be shown by studies on tic douloureux cases after section of the sensory roots. All sensation in the head is not abolished. These people have deep sensation.

I have at present a patient who has complete anesthesia over the face as a result of a posterior root section for trigeminal neuralgia, and yet she has to have her posterior sinuses cocainized to wash out the sphenoid. I know that cocaine in the sphenoid sinus will relieve some headache, but whether that is from the great superficial petrosal is a little hard to say.

The microscopic picture of the great superficial petrosal nerve would seem to show that it is a sensory nerve. I am willing to grant the sphenopalatine ganglion is entirely sympathetic.

DR. PERRY G. GOLDSMITH: Dr. Wilson, about 26 years ago, had a very interesting article which he was good enough to send me. Any statements he makes upon the innervation of the drumhead must be accepted unquestionably.

In tuberculosis of the larynx or any malignancy, it is not uncommon in the advanced stages to have pain in the ear and that pain is usually a bad symptom.

I am not going to enter into the question of the sphenopalatine ganglion. I think it is an enormously overrated thing and I hope it is buried.

Some Observations on Facial Palsy. Dr. James A. Babbitt.

Major progress in overcoming VIIth nerve palsy has occurred in the past 40 years. There seem to be four principal phases: 1. Research in the nature and physiology of nerve regeneration; 2. the period of anastomosis of distal segment with other motor trunk; 3. temporal muscle and fascial transplants to relieve deformity; 4. decompression and substitution of transplanted nerve in the Fallopian canal. Two cases of facial palsy of about 25 years standing are discussed; and the results given in one, following temporal muscle transplant in postauricular defect, and the other of temporal and fascia transplant to the region of eye and mouth and supplemented by fascia lata transplant (Dr. Ivy). These are illustrated by moving pictures. Conclusions are aided by findings of Ballance; Duel, Greenman and Fogliotti.

DR. JOHN R. PAGE: Dr. Babbitt states in his paper that, according to Cushing, the Ist nerve anastomosis was performed by Faure in 1898. I am sure that he will find in the article by Sir Charles Ballance and Arthur Duel, published in the Archives of Otolaryngology, January, 1932, that Sir Charles Ballance united the facial nerve end to end with the spinal accessory in 1895.

In his first case, the essayist says, the improvement in the facial muscles must have been due to the large pedicled graft of temporal muscle that was used to fill the mastoid cavity and which, fortunately for the facial muscles, extended 2 cm. below the lobe of the ear. It is almost fantastic to believe that innervation of the muscles of the face came from the lump of temporal muscle that was used to fill the mastoid cavity, but such must certainly have been the case. In Rucker's article on Surgical Treatment of Facial Paralysis, printed in German in 1933, he says that the experiments of Erlacher showed that the motor nerve fibres of implanted muscle grew into the injured muscle and form a new innervation, and that on the basis of these findings Rosenthal implanted broad flaps of the temporal muscle and masseter into the muscles of the eye, cheek and angle of the mouth. He furthermore states that muscular neurotization presents distinct advantages as compared with neuroplastic procedures. In Dr. Babbitt's first case, however, no strips of muscle lay in contact with any of the facial muscles themselves, but the end of a lump of muscle supplied by the Vth nerve extended from the mastoid cavity and lay only somewhere near the degenerated branches of the VIIth nerve in the region of the parotid. In the second case, the idea of taking strips of fascia lata and making swings of them to support the sagging muscles of the affected side is particularly useful in conjunction with muscle nerve grafts in cases of long standing paralysis to oppose the pull of the active muscles on the opposite side and give better opportunity for the muscle nerve graft to take effect.

According to the abstract of Rucker's article referred to, among the older attempts of myoplastic work were those of Gomoiu, who used the sternocleidomastoid, and Jiam, who employed the masseter. Lexer, likewise, used the masseter for a flap split in two tongues, one being brought above and one below the angle of the mouth, and attached through a small incision at the angle with the corner of the mouth drawn up in an overcorrection that he deemed necessary. In cases of paralysis of the upper branches of the facial

nerve he used also a flap from the temporal muscle and drew it through the undermined skin to be anchored by sutures under the paralyzed eyelid.

The interesting and encouraging statement in the literature is the reference to the experiments of Erlacher, which are said to show that the motor nerve fibres of implanted muscles grow into the paralyzed muscle and form a new innervation of it. Today we have had shown us an example of an innervation of paralyzed muscles, not from a strip of live muscle in contact with them, but from muscle that was barely in contact with the trunk of the degenerated nerve that supplied them.

DR. JOSEPH C. BECK: The first two cases shown here are very encouraging, particularly the first one. It would have been very interesting to have had the neurological diagnosis, that is, the reaction to degeneration. In these cases, Faure, the first man who operated on facial paralysis, according to my knowledge, although I do not wish to dispute the latter investigation, said that such an association was not possible after a reaction of degeneration, that is to say, after a true fibrosis of the paralyzed muscle was present; therefore, I would like to have that point cleared up by Dr. Babbitt.

During my years in practice I have operated 17 cases of facial paralysis by anastomosis, first by the spinal accessory as first described by the late Dr. John B. Murphy, of Chicago. That method was, of course, discarded because of the associated movement of the face with the movement of the arm. The facial-hypoglossal anastomosis brought end to end and end to side gives a much better result. A good many years ago I reported in the Annals of Otology, Rhinology and Laryngology a case successfully operated by end to side anastomosis of the facial-hypoglossal nerves so that the tongue was not affected.

The operation which apparently is not being discussed here today, namely, that of the late Dr. Duel and Sir Ballance, I think should at least be mentioned in an encouraging manner.

Regarding the work of transplanting fascia and muscle, I have had considerable experience. The external masseter muscle split and the free end brought into contact with the angle of the mouth gives the best result of any that I have tried. The temporal muscle when employed is thinned out, and it is very difficult to perform the operation on that account.

DR. EDMUND P. FOWLER, JR.: The movies of Dr. Babbitt's first case are typical of a VIIth nerve repair. I would like to ask whether the movement in the face of this boy was produced in an effort to move his face, as for a smile, or by biting his teeth together. If it was not produced by biting his teeth together (that is a movement which is activated by the Vth nerve) I would suggest that the return of his facial movement was due to releasing the neurons of the VIIth nerve in the mastoid wound; that is, it was the revision of the mastoid and the removal of the neuroma in it which is responsible for this boy's return of facial function and not the temporal muscle transplant.

DR. JAMES A. BABBITT: Any priority recognition due Sir Charles Ballance in the nerve anastomosis operation should be adjusted. There seems to have been some confusion in the literature as to chronology.

In answer to Dr. Beck I would say that the neurologic reports were quite definite. I am not entirely clear as to Dr. Fowler's suggested explanation, and cannot state definitely as to patient's mouth closure on producing facial movements. He made these as active as possible in answer to our request.

(To be continued in a succeeding issue.)

BOOK REVIEWS.

The New Acoustics. A Survey of Modern Development in Acoustical Engineering. By N. W. McLachlan, D.Sc. (Engineering) London, M.I.E.E. 166 pages with index and 100 illustrations. New York: Oxford University Press, 114 Fifth Avenue. Price $2.75. 1936.

This volume might well be called a Handbook on Acoustics. It presents both the theory and the practical application of most of the well known principles of acoustics as well as a history of the important developments in this field. The author is able to present his material in such a fashion that it is comprehensive to both the layman and the trained engineer. A considerable portion is devoted to the analysis of sounds, the behavior of the ear, and to hearing aids. Inasmuch as the author has spent the greater part of his lifetime in the study and development of acoustical devices, he is unusually well qualified to cover the entire gamut of acoustical science. The book is extremely broad in scope but sufficiently detailed in certain sections, e. g., the chapters on microphone developments and that on the accurate measurement of frequency, to render it a valuable reference for a design engineer or an instructor in Acoustics. The book begins with the "Old Acoustics" and "The Old Classical Theory" and ends with the "Measurement of Sound-Absorption Coefficients." The last chapter is rather brief and leaves much unsaid. Of particular interest to engineers are the diagrams, electrical analogues of mechanical systems, and equivalent circuit figures. The book contains more than a hundred such diagrams and pictures of modern devices and sound studios. J. A.

Allergy of the Nose and Paranasal Sinuses. A monograph on the subject of Allergy as Related to Otolaryngology. By French K. Hansel, M.D., Assistant Professor of Clinical Otolaryngology, Washington University School of Medicine; Fellow of the Association for the Study of Allergy, the Association of Resident and Ex-Resident Physicians of the Mayo Clinic; the American Laryngological, Rhinological and Otological Society, and the American Academy of Ophthalmology and Otolaryngology. With 58 text illustrations and three color plates. St. Louis: C. V. Mosby Co., 1936.

Allergy, both in its general and special phases, has been developed as a highly specialized field of medicine, and the author of this volume has presented an up-to-date epitome of all phases of this important problem. Not only are the general allergic manifestations of the patient, as they are presented to the internist, the gastroenterologist and the dermatologist given careful consideration, but the laryngologist is here offered a most useful digest of all subject-matter that pertains to the association of his clinical and therapeutic practice in its wide domain.

Pollen allergies, food reactions, dermic tests, the physiological character of the mucosa of the nose and upper respiratory tract, reflexes, neuroses and the long array of causative factors in allergic conditions of the body are given due attention.

Bio-Chemistry of the secretions, cellular reactions of the tissues, immunization, histopathology, as they relate to the nose and paranasal sinuses, constitute important chapters of this book.

To each chapter is added a complete bibliography that enables the reader to follow up his subject-matter minutely.

It is a most comprehensive volume on this important and often baffling subject-matter that has as yet come to our notice and should be included in the reference library of every progressive reader in the profession.

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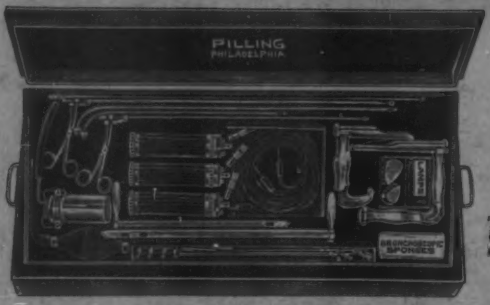

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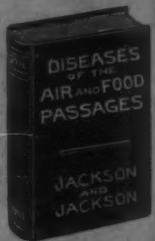

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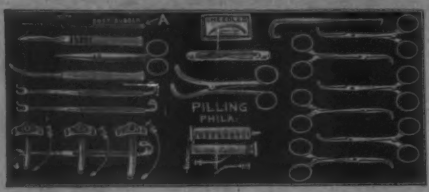

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